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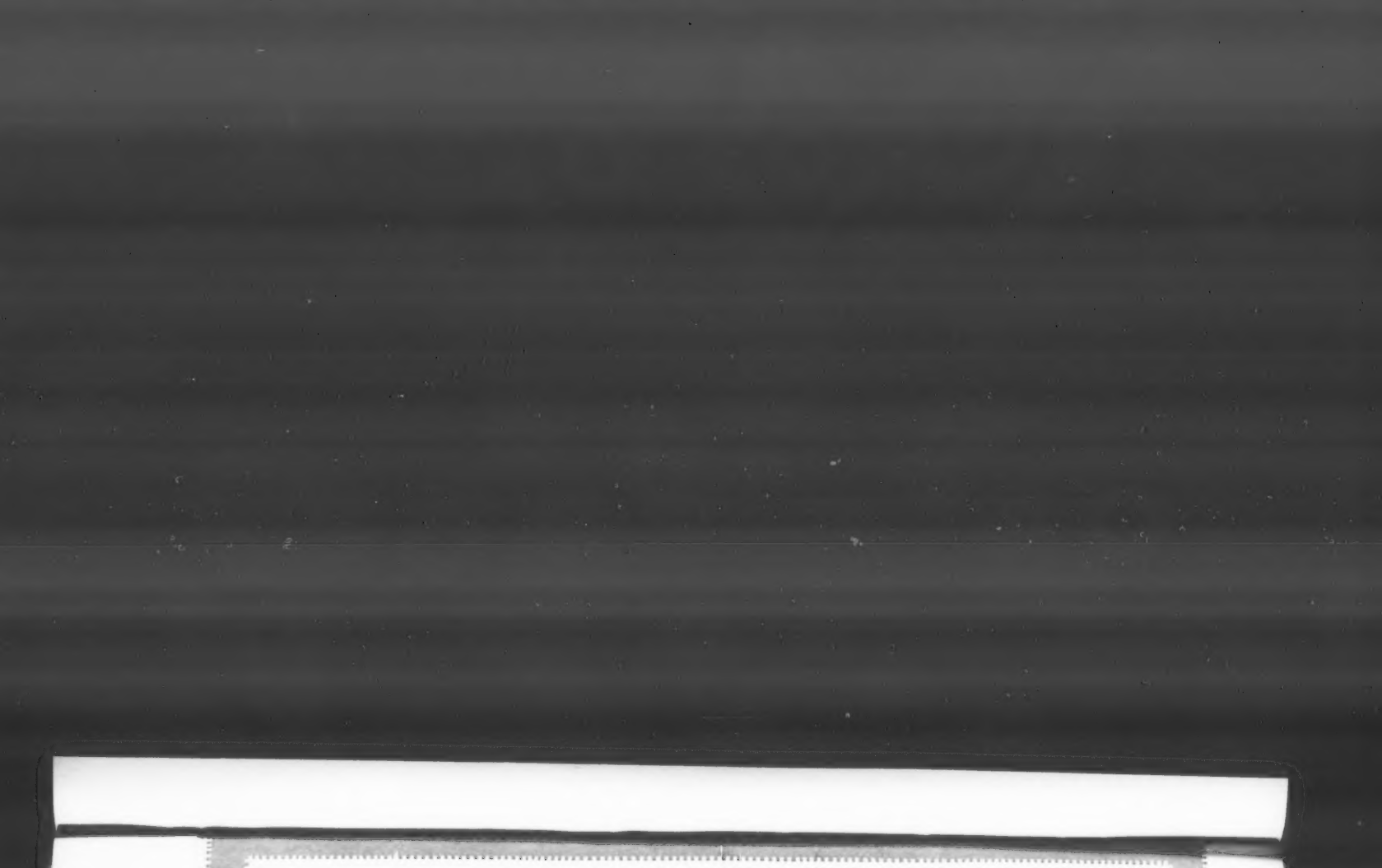

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THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES

MARCH, 1916.

ORIGINAL ARTICLES

THE CAUSES OF DEATH IN DIABETES.[1]

By Elliott P. Joslin, M.D.,

BOSTON.

(From the Nutrition Laboratory of the Carnegie Institution of Washington in Boston, and the Department of Medicine of the Harvard Medical School.)

With the lapse of years the conviction has grown upon me that the life of a diabetic patient might be prolonged and that coma was an avoidable accident, and yet I know of no cure for diabetes and I claim no patient cured. The results of the investigations conducted with Professor Benedict at the Carnegie Laboratory upon the utilization of carbohydrate in diabetes, and reported here last spring,[2] greatly strengthened that belief, for they proved that even the severest diabetic retained some power to burn carbohydrate. Then, too, in these last few months I have been unable to find any uncomplicated case of diabetes of long or short duration which has failed ultimately to show at least a small positive carbohydrate tolerance. The simultaneous observations of the Russell Sage Laboratory at Bellevue Hospital and the Carnegie Nutrition Laboratory showing that the respiratory quotients of severe diabetic patients rise following a prolonged fast, although not fully explained, are encouraging. And, finally, I must acknowledge that the fact that under simple prolonged fasting treatment no death from diabetes occurred among my patients at the New England Deaconess' Hospital for 350 days, has deepened the impression above expressed.

[1] Delivered at New York Academy of Medicine, December 21, 1915.

[2] Harvey Lecture, Delivered March 13, 1915. Also published in Arch. Int. Med., 1915, xvi, 693.

Enthusiasm governs the present, but facts the past. Consequently, I have endeavored to secure the end-results of all the diabetic patients I have seen in private practice from the first case of my student days in 1894 to November 18, 1915. Of these 945 cases, 921, or 97.5 per cent., have been traced, and out of this number 425 have died. The actual causes of death are known to me in all but 5 cases. It is true that many of the patients were seen by me but once, yet this by no means detracts from their interest.

TABLE I.—CAUSES OF DEATH OF DIABETIC PATIENTS SEEN IN PRIVATE PRACTICE ONE OR MORE TIMES FROM 1894 TO DECEMBER 18, 1915.

		Number.	Deaths in hospital.	Deaths outside hospital.
A. Deaths Without Coma		147	2	145
I. Miscellaneous		16	0	16
Pernicious anemia	1			
Old age	2			
Diabetes	8			
Cirrhosis	2			
Suicide	2			
Drowned	1			
II. Cancer		17	1	16 also 1 with coma.
Face	1			
Esophagus	1			
Stomach	4			
Rectum	1			
Liver	6			
Bladder	2			
Bones (sarcoma)	1			
Seat of growth unknown	1			
III. Pulmonary tuberculosis		16	1	15 also 5 with coma and 2 with infections.
IV. Cardiorenal and vascular		62	0	62
Cardiac	28			
Bright's	14			
Cerebral hemorrhage	14			
Arteriosclerosis	6			
V. Infections		36	0	36
Tonsillitis	11			
Pneumonia	15			
Influenza	3			
Gall-stones	1			
Septic and gangrenous legs	9			
Carbuncles	4			
Acute appendicitis	2			
Erysipelas	1			
B. Deaths With Coma		273	15	258

A. Deaths Without Coma. The causes of death of these patients will be discussed under six headings, into the first of which I will place the miscellaneous and uncommon causes.

I. *Miscellaneous.* In this group fall 1 death of pernicious anemia, 2 of old age (aged seventy-eight and eighty-six), 8 of which I can secure no other details than a death report signed diabetes, 2 of cirrhosis of the liver, 1 was drowned, and 2 committed suicide.

36 C 1

29 My 44

Of the latter, one was mentally unbalanced and the other took her life after becoming pregnant a second time soon after an abortion. Today I should not advise an abortion unless vigorous and prolonged treatment failed to render the urine sugar- and acid-free.

II. *Cancer.* Seventeen of the patients died of cancer. The primary growths were in the face, esophagus, stomach, rectum, liver, bladder, and I include 1 case of probably sarcoma of the pelvic bones. The average age at death of these patients was sixty-five years. Cancer apparently developed after the diabetes in 8 cases, and may have developed before or coincident with the diabetes in 7 others. Incidentally, I would say that 2 cases of cancer which have been operated upon are alive, 1 of the bladder and 1 of the uterus. A patient with cancer of the bladder died suddenly of pulmonary embolism nine days after its removal and another with recurrence in the abdominal scar.

III. *Pulmonary Tuberculosis.* Pulmonary tuberculosis was responsible for the death of 16 cases. The medical profession has altogether too pessimistic a view about this complication. When the treatment of the diabetes is faithfully carried out these patients do quite well. The trouble in the past has been that consumption was usually advanced when diagnosed. In a diabetic, temperature, pulse, and respiration give no clue to the diagnosis, and the loss of weight is attributed to the diabetes. One case of tuberculosis died under my very eyes in the hospital without my suspecting what proved to be extensive lesions. That this mistake is common I am convinced because, with my mistake in mind, a few months later, in one of the very best hospitals, I was able to point out advanced but also unsuspected tuberculosis in a diabetic. The detection of tuberculosis in diabetics is aided by a careful study of the family history and an *x*-ray examination.

IV. *Cardiorenal and Vascular.* Cardiorenal and vascular changes, uncomplicated by coma, caused the death of 62 cases. Of these 28 died of heart disease, in 4 instances suddenly with angina pectoris, 13 died of chronic nephritis, 14 from a cerebral hemorrhage, 6 of general arteriosclerosis, and 1 of pulmonary edema. The average age at death of these patients was sixty-four years. If the methods we employ in the treatment of these same arteriosclerotic conditions in non-diabetic patients are correct, it is difficult to see how modern treatment with restricted instead of forced feeding can fail to prolong the life of these individuals.

V. *Infections.* The advent of an infection lowers the tolerance of a diabetic for carbohydrate and thus increases the severity of the disease. This is an old and reliable clinical fact. It has lately been emphasized by the Chicago School. Case 813, eight years old, sugar-free for eighteen days, developed tonsillitis, and without change of diet a positive carbohydrate balance of 50 grams dropped to 14 grams, with reappearance of acidosis. Recent experiences

with fasting treatment show that in the presence of an infection a diabetic becomes sugar-free very slowly. An infection is an additional load for the diabetic to carry, and to it he often succumbs. If those cases are excluded in which coma was an element the number of deaths from infections is comparatively small, for there were but 36 cases. Of general infections, pneumonia heads the list with 15 deaths and influenza claims 3. Of local infections, septic and gangrenous legs account for 9, carbuncles for 4, and acute fulminating appendicitis for 2.

Thus far we have accounted for 147 deaths. Of this number it is problematical how many could be saved today. Perhaps one death from suicide could be prevented, a little could be accomplished for the cancer cases, the diseases might be arrested in a few and life prolonged in most of the tuberculous, but how effective modern treatment would be upon cardiorenal and vascular conditions in diabetes time alone will tell. But the majority of my diabetics died from another cause, and now let us face it.

B. Deaths with Coma. Coma was fatal to 273 of my cases (64.24 per cent.), thus causing two-thirds of the deaths. During the last fourteen years there have been treated under my personal supervision in hospitals, 273 cases of diabetes, of which 15 have died of coma, making my total deaths in hospitals for this period 17. For many years my attitude toward coma was to consider it the culmination of the diabetes, and after the death of the comatose patient I could honestly unite with the family, the physician, and the nurse in the feeling that no more could have been done. But for me this comfortable creed has passed, and in its place has come the opinion that coma by no means represents the culmination of the disease, that it is not a justifiable accident, and though not in all, yet in most cases it is avoidable. My conception of coma in diabetes cannot better be expressed than by quoting from the charming essay of Moynihan on "Inaugural Symptoms in Abdominal Emergencies with Especial Reference to Duodenal Ulcer," only I shall change the word "ulcer" and the phrase "in the abdomen" to "diabetes," and the word "perforation" to "coma."

With the wording thus changed the text would read: "It is in dealing with the acute catastrophes occurring in *diabetes* that we shall probably derive the most instant and striking advantage from an attentive study of inaugural symptoms. To take a specific example, the onset of *coma* in *diabetes*. But let me first say that a catastrophe of this kind is almost always capable of being forestalled. Though the onset of *coma* in *diabetes* is acute the *diabetes* itself is chronic. It is a disease that has existed for months or years and it has given, in almost every instance, not only sustained evidence of its existence, but a recent warning that the pathological processes engaged in it were becoming more acute. The warning, however, is commonly ignored, because the significance and import-

ance of it are not understood, and accordingly a disaster is precipitated. There are few catastrophes occurring in diabetes that are veritably acute."

Bearing this point of view in mind, may I analyze for you the deaths from coma which have occurred in my practice?

1. *Ether Anesthesia.* Case No. 729, a severe diabetic, three months after her last visit to me, without my knowledge, was taken to a dentist's office, given ether by her physician, and all her teeth extracted. This was on a Monday. She was taken home, became unconscious Wednesday, and died on Friday. I cannot force myself to believe that her death represented the culmination of diabetes, or was even accidental. Case No. 348, before an operation for removal of a prostate, was free from acid and sugar and tolerated 20 grams of carbohydrate. After light etherization followed by fasting, 33 and 41 grams of sugar appeared in the urine on the second and third day of the fast respectively and the ammonia was 3.3 grams. It is true that he recovered, just as all but 6 of 26 patients under my supervision who have had major operations have also done, but the ether lowered his tolerance and made his diabetes temporarily worse. This does not mean that diabetics should not be operated upon, but it demonstrates that ether anesthesia is a burden which a light case of diabetes may easily bear, which may change a moderate to a severe case, and to a severe case may be fatal.

2. *Impaired Kidneys.* Diabetic patients with vulnerable kidneys are peculiarly susceptible to coma because the power of elimination of acid bodies is impaired. You all will recall cases of Bright's disease in diabetes in which an apparently mild acidosis preceded coma. Years ago, Goodall[3] and I pointed out that acidosis was much more easily tolerated by the young than the old diabetics, and this can well be attributed to the deficient kidneys of the latter. Few could void on the verge of coma the five or more liters of urine which with the old alkaline treatment was necessary for recovery, and is recorded to have taken place in those patients who did recover. Coma by no means is confined to the young: thus the percentage of coma in my fatal cases under fifty-one was 67 per cent. and above that age 33 per cent. I could specify several instances of this type and in this group might well be classed a few of the cases of death in pregnant women.

3. *Infections.* Already attention has been called to the fact that an infectious process renders the diabetes more severe. A considerable percentage of the cases of coma occurred in connection with either general or local infectious processes. It not infrequently happens that the infection is not recognized. Better statistics

[3] The Clinical Value of the Estimation of Ammonia in Diabetes, Boston Med. and Surg. Jour., 1908, clviii, 646.

upon this point and in general about the circumstances attending coma should be accumulated. Thus, Case No. 836, seen in consultation one evening, was found to be in partial coma, but I was able to demonstrate to the physician a membrane in the throat, and three hours after the patient's death the following morning, the Board of Health reported a positive culture for diphtheria.

But what I consider of far more importance is the number of procrastinating cases of mild infections in mild diabetics, chiefly in their lower extremities, which frequently prove fatal. The youngest case of sepsis or gangrene of the legs in a diabetic in my personal experience has been fifty years. In other words, these conditions develop at a time of life when diabetes is mild, and why should they so frequently be fatal? Consider with what these mild cases of diabetes have to contend. Handicapped by a lingering infection, which only too often is allowed to continue for months, with kidneys less efficient for throwing off the acidosis attack, deprived of exercise—that recently proven stimulus to sugar consumption (for whoever heard of a poor, old, gangrenous diabetic taking exercise)—these pitiful patients frequently meet a fourth enemy in ether anesthesia; and is it any wonder that a formerly innocent disease becomes virulent and the victim dies of coma? There is no doubt in my mind but that if such cases had been treated vigorously, even with the dietetic methods of a few years ago, a large percentage of the legs amputated might have been saved. In fact, Dr. Stettin has most ably demonstrated this. If one will read his paper it will be seen that his success in these cases was due to two factors: (1) that his patients were given the very best medical treatment of the time, and (2) that they had the advantage of expert surgical care. It is known that if a diabetic patient has gall-stones to be removed he instantly commands the services of the leading surgeon on the senior staff; but if a diabetic patient has a sore toe there is no house officer too young to dress it, until a few weeks later, if the patient survives that long, the surgeon in the amphitheatre amputates the thigh. I recommend to your careful perusal Dr. Stettin's article.[4] Any success that I have had with surgical patients in diabetes has been due to the fact that I know no surgery, and learned that never-to-be-forgotten lesson twenty-one years ago; and, further, that no matter how trivial the ailment I have secured the very best surgical skill for my patients.

4. *Mental Excitement.* An occasional case of coma is precipitated in a severe diabetic by mental excitement. A violent fit of anger in one of the hospital patients, already in a precarious condition, was accompanied by vomiting and inability to retain liquids. Coma soon appeared, and this case I know to have been duplicated by another outside the hospital. A patient with exophthalmic goitre went into coma with far more ease than is the rule.

[4] Jour. Amer. Med. Assn. 1913, lx, 1126.

5. *The Influence of a Fat-Protein Diet or Fat Poisoning.* The explanations of coma thus far given account for many deaths, but by no means for the majority of deaths from coma in diabetes. In these fourteen years at the hospital 9 cases of coma have occurred under my care which could not thus be explained. It is easy to say that the patients die soon after admission, but I cannot get around the thought that if a patient reaches the hospital alive I am responsible for his departure from the hospital alive, too. I will partly excuse myself for the death of one patient five hours after entrance and for a child of two years, who was in coma in ten hours, but I will not plead any excuse for the other 7 deaths. A study of these patients shows none of the factors hitherto mentioned as predisposing to coma, but there is one factor which is common to all, namely, the diet consisted largely of fat and protein with little carbohydrate, or just prior to entrance or after entrace an excessive quantity of fat had been given. Diabetic patients will live untreated for many years without the appearance of coma. They suffer from complication after complication. They are tormented with sepsis, neuritic pains, and pruritus; yet they still live. Their diet is atrocious. Along comes an enthusiastic young doctor —and years ago I could easily have been classed in that group— and presto! change! fat is increased, carbohydrate diminished, and the patient goes into coma. Out of carbohydrate it is impossible to form the acid bodies. When, therefore, carbohydrate is suddenly replaced with fat we deliberately furnish our diabetic patients with material which though it acts partly as a food, acts far more as a poison. At a recent meeting upon acidosis in children, Dr. Jacobi really struck a keynote. He said that prevention is the treatment of acidosis for children, and that those susceptible to acidosis ought not to have fat. What he said for children holds for diabetics. Diabetic patients need fat—it forms the chief constituent of their diet—but they must not be poisoned with it; they must be gradually accustomed to it.

The treatment of acidosis in the past has been unsatisfactory. Like typhoid fever, it should be prevented—not treated—and in uncomplicated cases this can be done. Although 90 per cent. of our diabetic patients will quickly and safely become sugar-free by simple fasting, for the remaining 10 per cent., which would include cases already showing acid poisoning, elderly patients and diabetics with infections, I suggest that prior to giving any fasting treatment a possible acidosis be anticipated by taking away the cause— namely, by the absolute exclusion of fat from the diet, but otherwise make no change in the diet or in the habits of the patient. After two days, or longer if desired, omit protein—another, though lesser contributing factor to acidosis—and thereupon daily halve the carbohydrate hitherto unchanged in the diet, until 10 grams are reached, and then proceed with routine fasting treatment. I would

welcome information as to whether such treatment fails to prevent the appearance of acidosis or to lessen an acidosis already existing. Your aid is solicited, for you must remember that under plain fasting 350 days intervened without a death in the hospital, and if this preliminary treatment to fasting is at all advantageous it will require some time to test its efficacy.

6. *Rapid Loss of Body Fluid.* Vomiting at the onset of coma usually presages death, because the patient is deprived of fluid with which to eliminate acids. Body liquids are so needed that the body in the course of coma becomes obviously dry. This is really only another cause for defective elimination.

But I have been interested for many years in the importance of salt for a diabetic. Recently, too, my attention was drawn by a patient to the fact that in another hospital he lost thirteen pounds during the first four days of fasting treatment. Inquiry developed that he was given, during this period, only water and alcohol. Had he been given broths or some mineral water, suffice it to say that during fasting, even for a week, he would probably have lost little or no weight. This is important, because patients prior to coma frequently lose weight rapidly. Thus in the presence of multiple carbuncles and septicemia one of my patients lost thirty-six pounds in eleven days preceding his death. Years ago in seeking for the cause of edema in a severe diabetic patient who was taking sodium bicarbonate I withdrew sodium chloride from his diet. Prompt loss of weight followed and symptoms of coma appeared. Since then I have been careful not to restrict salt, and I can well understand the opinion of Hodgson, that water rich in salts is really helpful to diabetic patients. On the other hand I would point out that large doses of sodium bicarbonate frequently are detrimental to a patient with threatening coma. How many patients have we had recover who took 20 teaspoonfuls of soda a day and a tablespoonful of whisky every hour and a half? For the last four months not one of my patients has been given a teaspoonful of sodium bicarbonate, and so far I have seen nothing but good results. I prefer to follow Dr. Jacobi's advice and prevent rather than to treat acidosis, and even when it appears I am inclined, though I hold myself ready to change my opinion any moment, to accept the advice of another master of medicine in New York, Prof. Herter, who in 1901 told me he preferred to let his diabetic patients neutralize their acidosis with their own ammonia rather than to give them sodium bicarbonate.

While seeking out the fatal cases of diabetes the number of living patients was ascertained. For comparison I have arranged both groups in decades and completed the average length of life of the fatal cases and the average duration of life of the living cases up to December 1, 1915. It is obvious that among the living cases will be found many in whom the disease is of recent origin, and for

this reason the duration of life of the living cases appears far too low in the table. In another place the living cases will be analyzed more in detail. One of the chief reasons for publishing these statistics which cover the period 1894 to 1916 is the criterion which the table will afford for the estimation of the value of treatment by modern methods.

TABLE II.—DURATION OF LIFE OF 408 FATAL AND 490 LIVING CASES OF DIABETES, DECEMBER 1, 1915.

Age at onset. years.	Number of cases.		Average duration in years.	
	Fatal.	Living.	Fatal.	Living.
0 to 10	33	9	2.06	4.44
11 to 20	48	27	2.79	2.70
21 to 30	40	50	3.30	4.90
31 to 40	53	71	4.43	6.12
41 to 50	71	146	6.08	7.04
51 to 60	97	120	6.63	6.29
61 to 70	52	55	6.00	5.38
71 to 80	14	11	3.71	4.45
81 to 90	0	1	....	0.33

LINITIS PLASTICA.

By Joseph Sailer, M.D.,
PHILADELPHIA.

Linitis plastica is a condition in which the wall of the stomach becomes greatly thickened, loses its motility, suffers a marked diminution in its functional activity and as a result leads to death by starvation. The terms of this condition are numerous and all more or less descriptive, leather-bottle stomach, Schrumpf Magen, hypertrophic gastritis (Cruveilhier), chronic indurative sclerosis of the pylorus, cirrhosis of the stomach, linitis plastica (Brinton), fibroid induration, gastric sclerosis are sufficiently indicative of the fact that the wall of the stomach is thickened and hard. Although Cruveilhier and Andral and others had called attention to the existence of this condition earlier, William Brinton was the first to regard it seriously as an independent disease. He devotes a chapter to it in his lecture on "Diseases of the Stomach," and describes two forms, that of contraction and the rarer form of dilatation, and expresses the belief that in a partial form the condition is fairly common, a belief that further experience has not confirmed. He cites no case but must personally have seen several, and for various reasons, given chiefly in a foot note, prefers the name linitis plastica to any other designation. The next important article was that of Hanot, in 1882, who reported one case and analyzed the literature. This was followed in 1893 by the article

of Tilger, who also reported a case and collected all the certain cases previously reported in the literature. His own case occurred in a woman, aged thirty-five years, who had a dilated stomach and an obstruction at the pylorus. An attempt to operate was rather futile on account of the desperate condition of the patient, and she died on the following day. Codivilla collected 23 cases, all of which died and in which the diagnosis was confirmed by autopsy. Of these 13 were men and 10 were women, and the ages ranged pretty regularly from twenty to seventy. He denies very earnestly a carcinomatous nature of the condition. In a postscript he mentions a case reported in which a correct diagnosis was made during life, and digital devulsion twice practised with cure, and Hemmeter, in his text-book, which contains a very excellent article on this subject, reports 2 fatal cases and mentions the fact that he has also seen two others. In 1907 von Sury reported a case from the Pathological Anatomical Institute of the University of Basle with very careful histological study. He defines the condition as follows: (1) a primary more or less extensive increase in the thickness of the wall of the stomach as a result of new-formed connective-tissue, particularly in the submucosa and subserosa; (2) diminution in the volume of the stomach as a result of secondary contraction. This is usually the result of an existing or completed inflammation of the wall of the stomach. He also gives a brief clinical picture of the disease, of which the most interesting features are the frequency of ascites and the fact that a tumor can usually, sooner or later, be felt in the epigastrium. The duration is from three to five years. It occurs in early as well as late life and there are neither metastases nor hematemesis. All these conditions distinguish the condition from carcinoma. The surgical treatment is the most important. He is rather inclined to group it with fibrous polyserositis, either the form described by Pick or the iced liver of Curschmann. One hundred and seventy-two references to the literature, the largest collection hitherto made, are appended to this article. In 1898 Boas reported three characteristic cases of this condition all of which had been cured by gastro-enterostomy. They were all of the obstructive type. It must be admitted that in none of these cases was the subsequent history known for a sufficiently long time to exclude the possibility of a carcinomatous change. Quenu, in 1908, reported 2 cases of gastrectomy for this condition. The man lived two years and then died of hemorrhagic pleurisy, probably carcinomatous. The woman lived two and a half years and died of a local return. In both cases the histological examination indicated the carcinomatous nature of the process. Munter in discussing four cases of this condition reached a conclusion that there are several forms of chronic gastritis. These forms are first linitis plastica. This is characterized by marked thickening of the gastric wall due to proliferation of the connective-tissue.

Thickening usually begins at the pylorus and leads gradually to contraction with partial stenosis and possibly in the end, if the patient survives long enough, to complete stenosis. There is some tendency to the formation of adhesions to the surrounding organs, and in all probability the lymph glands adjacent to the stomach frequently become enlarged, but there is no tendency to metastases. The cachexia is solely due to starvation, and the prognosis appears to be favorable if the obstruction is relieved by gastro-enterostomy. Curiously enough there is good reason now to suppose that after the gastro-enterostomy some degree of retrograde change takes place and the stomach to a certain extent returns to a normal condition. Second, a diffuse carcinoma of the wall. For some reason that is not rendered clear by a study of the literature it seems to be the opinion of many surgeons that this is the only type of the disease. It is unfortunate that this should be so, as it leads to a pessimistic prognosis which is invariably justified if no operative interference is undertaken. This is characterized by thickness of the wall, the histological picture of which is probably not greatly unlike that of pure linitis. I have had the opportunity of studying one case of carcinomatous linitis gastrica. At least I was under the impression at the time that this was the correct diagnosis. The patient, a negro, was brought to my service at the Polyclinic Hospital some years ago with symptoms of obstruction at the cardia. This was diagnosed carcinoma without definite proof, and gastrostomy was advised. The wall of the stomach was found thick and leathery. Nevertheless, Dr. John B. Roberts succeeded in making a satisfactory gastric fistula through which the patient was fed, and he gained weight. The power of swallowing through the esophagus returned, and the patient, who was a caterer, left the hospital and indulged his appetite pretty freely. He was brought back forty-eight hours after his discharge suffering with the symptoms of peritonitis, to which he succumbed in a very short time. No autopsy was permitted. The clinical picture of carcinomatous sclerotic gastritis and linitis plastica does not differ in the beginning. There is no satisfactory way of making a differential diagnosis excepting by an incision and histological study of portions of the gastric wall, and for reasons that I shall consider later, even this may be unsatisfactory. The real differentiation is made not by the more rapid course and greater cachexia, because these after all are quantitative and untrustworthy, but by the occurrenc of carcinomatous metastases to other organs, particularly the liver.

The third condition is syphilis. If the other signs of sclerotic gastritis are present and the patient has a positive Wassermann reaction it is justifiable to use antisyphilitic medication actively with the hope of benefiting the condition. As far as I know there are no cases on record of this type of the disease that have actually been cured by antisyphilitic treatment. Finally, it has been sug-

gested that chronic sclerotic gastritis may be a part manifestation of a multiple serositis. These are probably cases characterized by ascites that rapidly reaccumulate, and it is doubtful whether the gastric symptoms ever become very definite. In the only one case of this kind that I saw, a young man, aged twenty-seven years, who had obliterative pericarditis, chronic pleuritis with reaccumulation of pleural fluid and chronic ascites with rapid accumulation of ascitic fluid, the gastric wall was found greatly thickened. At the autopsy this thickening was found to be due to a curious hyaline change in the serous membrane similar to that found over the lung, liver, and spleen, but there had been no gastric symptoms of importance during the brief course of the disease.

The prognosis without treatment is hopeless. No medical treatment is known. Therefore the only hope lies in some efficient form of surgical interference. In 1908 von Eiselsberg reported a case upon which he had operated in 1902. The patient, a woman, aged forty-one years, had been ill for about three years with vomiting and loss of weight. At the first operation the stomach was found to be thickened and adherent to the surrounding tissues. Nothing was done excepting to free a few adhesions. At the second operation a jejunostomy by the Witzel method was performed. For one year she was fed almost exclusively through the fistula with liquid food. She regained her former weight of 72 kgms. Later she increased still more in weight and was able to take a certain amount of food by the mouth. Six years after operation she was still in good health, had married again, and weighed more than at any time before the operation.

The direct diagnosis depends (1) upon the clinical signs, (2) on the *x*-ray findings, and (3) on a possible exploratory laparotomy. None of the clinical signs are really characteristic, and upon them alone I do not believe that a positive diagnosis can be made. A diagnosis can be surmised, however, if the symptoms develop gradually; if there is evidence of decreasing capacity of the stomach, which might occur also in hour-glass stomach; if there is fulness and perhaps a firm, rounded mass in the epigastrium; if there is vomiting that often depends upon the character of the food ingested, so that the patient can often predict when it is likely to occur. These things may lead to a suspicion of a contracting wall. The *x*-ray picture is usually characteristic. The small irregular lumen, the irregularities remaining constant in successive pictures; the contraction of the lower portion, the expansion of the fundus and, as occasionally seems to happen, the permanently patulous pylorus with a rapid flow of bismuth into the intestines, all point to this one condition. I have recently seen, however, a patient whose *x*-ray pictures seemed to present most of these features, and yet at an operation nodular sarcomatous thickenings were found in the gastric wall. The results obtained from exploratory laparotomy

are, of course, decisive. If there are adhesions between the stomach and surrounding structures it is only important to recollect that in the absence of metastases a diagnosis of carcinoma can not be made with certainty.

Souper has recently reported an interesting case correctly diagnosed, that was of the form associated with stenosis of the pylorus, and the patient three years after the operation of gastro-enterostomy was apparently perfectly well. As Souper very sensibly remarks, the possibility of carcinoma is probably not to be considered now. Dr. David Riesman recently reported a case, as yet unpublished, before the Musser Clinical Club, and the patient is still alive and in good health five years after a gastro-enterostomy. It seems to me clear, therefore, that not all of these cases can be carcinomatous, although the two cases of Quenu suggest that at least some of them are, and in this state of uncertainty it is important to treat every case as if it were benign. Bland-Sutton, in a recent excellent article on this subject, is somewhat undecided in his views and inclined to think that many of the cases are not malignant in nature. To my mind one of the most difficult tasks in morbid histology is the differentiation of a chronic sclerosing inflammation of the mucous membrane from carcinoma. The pictures presented by the two conditions are often similar to a puzzling degree, and I should be loath to reply upon the histological evidence of the stomach wall alone, unsupported by demonstrated epithelial metastases to the lymph glands or other organs.

The following case came under my observation and an opportunity was presented to make careful studies:

The patient is white, aged forty-six years, a physician. His previous history is unimportant. He has been successful in his professional work. Has been very fond of outdoor life, particularly as a hunter of big game, in the pursuit of which he has spent many weeks at a time. He has indulged moderately in alcohol and tobacco. On June 1, 1913, he was operated upon for bilateral hernia. The operation was entirely successful, the wound healed by first intention, but for some days following the operation there was intense pain in both groins. In July he noticed distinct loss of weight and suffered from pain immediately after eating, that frequently was associated with nausea. To relieve both the pain and the nausea he resorted to voluntary vomiting. In May, 1913, he weighed 178 pounds, approximately his weight for the previous ten years. In December of the same year he weighed 160 pounds. The physical examination at this time was practically negative. The thoracic organs were normal. The abdominal walls were well relaxed, facilitating palpation, but aside from the descending colon no organs were felt. There were no areas of tenderness. Loud gurgling was produced by pressure in the epigastrium. Deep pressure just above the umbilicus was slightly uncomfortable. There was no splash

in the stomach two hours after taking food (glass of milk). By auscultatory precussion the greater curvature of the stomach appeared to be at the level of the umbilicus. The nervous system was normal. An examination of the blood showed a moderate degree of anemia, 7000 white cells and a normal differential count. The blood-pressure, 116 and 84, was slightly below normal. The gastric contents showed no free hydrochloric acid and a total acidity

FIG. 1

of 38; no occult blood; a very large amount of mucus; no Oppler-Boas bacilli; no lactic acid. There was definite evidence of peptic digestion in the diluted contents by Mette's method. The urine was normal. A diagnosis was made of severe catarrhal gastritis, the patient placed upon a careful diet and given daily lavage. This produced great subjective improvement for ten days. Then he complained of a crawling sensation in the stomach, with nausea and

pain after eating. His weight had decreased four pounds. At this time the gastric contents obtained two hours after a mixed test breakfast gave a total acidity of 46; no free hydrochloric acid. Peptic digestion was present when the contents had been diluted with $\frac{1}{20}$ normal hydrochloric acid solution. There was no occult blood; no lactic acid; no Oppler-Boas bacilli; no sarcinæ, but a great deal of squamous epithelium. During this time the patient

FIG. 2

had been on hydrochloric acid, but the last examination still failed to give free hydrochloric acid, although the total acidity had risen to 68. There was no evidence of proteid digestion and no peptic activity in the filtered stomach contents, but when diluted with $\frac{1}{20}$ normal hydrochloric acid in proportion to 1 to 16, proteid digestion was fairly active. In this specimen for the first time a large amount of pus was observed. Numerous attempts were made to obtain

test meals, only a few of which were successful, because the stomach emptied itself so rapidly. All gave practically the same result excepting the variation in the amount of total acidity. Pus was constantly present. Occult blood never. Very often even fifteen minutes after the ingestion of food the tube failed to obtain any of the gastric contents. An attempt was made with an Einhorn tube on two occasions to obtain duodenal contents, but if they were obtained they corresponded in all respects to the gastric contents, and nothing was learned excepting that in the supposed duodenal contents trypsin and bile could not be demonstrated. Early in January, Dr. Miller, of the German Hospital, took two *x*-rays that were clear and definite (see Figs. 1 and 2). They showed that the lumen of the stomach was very much reduced in size, was distinctly irregular in outline, and a diagnosis of linitis plastica was made. Subsequently fluoroscopic studies were made by Dr. H. K. Pancoast at the University Hospital. These confirmed the findings of Dr. Miller, indicating greatly thickened gastric walls, and in the fluoroscope a most extraordinary rapidity of the gastric emptying was observed. This was not hypermotility due to excessive peristalsis, but rather to the fact that the bismuth flowed almost immediately through the rigid tube formed by the thickened stomach walls into the duodenum. The patient lost weight for a time until he reached a minimum of 139 net. Later assimulation seemed to improve, and at one time his weight increased to 149 pounds, but averaged approximately 145 pounds. There was otherwise no change in his condition, excepting that from time to time some tenderness could be obtained just to the right of the median line near the costal margin, and occasionally a mass could be felt which seemed to correspond to the rounded greater curvature of the stomach. This could be felt on deep inspiration and was not constantly present. In view of the pus it was suspected that some infectious process might be present in the wall of the stomach, and Dr. Damon R. Pfeiffer performed a series of agglutination tests with all the microörganisms that he happened to have in the pathological laboratory of the German Hospital, amounting to about twenty. These included all the ordinary pyogenic organisms, colon, typhoid, and various other strains of paratyphoid, but all were negative. Several hypodermic injections of theosinamin were made without definite result, although the patient complained of a distinct sensation in the region of the stomach, and also, somewhat unexpectedly, in the hernial operation wounds, immediately after the injection. There was no evidence of improvement, and they were discontinued. It was therefore decided to perform an exploratory operation, which had not been done earlier, partly because there was no evidence of obstruction in the gastro-intestinal tract and partly because the surgeon consultants had advised against it. However, they were persuaded. The operation was merely an inspection of the

abdominal viscera, which confirmed the clinical diagnosis, and the abdomen was then closed. The surgeons were very positive that the condition was cancerous. There was, however, in spite of careful search, no evidence of metastasis. There were no enlarged glands along the lesser curvature, but some masses were found around the greater curvature that were probably enlarged lymph glands. There were some very slight adhesions around the pylorus, which were separated, and the great omentum was adherent to the wall of the abdomen, apparently close to the sites of the operations for hernia. The patient made a prompt and satisfactory recovery from the operation. Six months later his condition had distinctly deteriorated. For a time he had felt better and had been able to eat nearly everything. His strength increased, although his weight did not go above 140 pounds. He was able to take long walks and even to go on shooting expeditions. Then after about five months he noticed that liquid food could not be taken with comfort, and this applied also to soft food such as fruit, light desserts, etc. There was a good deal of nausea and almost complete anorexia. His weight steadily decreased to about 132 pounds. He appeared cachectic and was incapable of much exertion without great fatigue. The physical examination showed only a more distinctly palpable mass in the epigastrium extending under the right rib. The *x*-rays showed distinct contraction of the lower portion of the stomach, the lumen was greatly reduced, and the thickness of the wall not very much increased. The condition appeared to be exceedingly desperate, and it was resolved to attempt a second operation with the idea that if a gastro-enterostomy could not be performed, and it was not our expectation that it could, at least a jejunostomy could be attempted in a hope that the brilliant results obtained by von Eiselsberg in his case would be reproduced. Dr. Finney, of Johns Hopkins, was consulted, and he believed that in view of the apparent hopelessness of the condition unless something were done, it would be worth while to make a trial. The patient accordingly went to Baltimore, and in October, 1914, Dr. Finney reopened the abdomen. He found that the stomach was hard, contracted, and bound by firm adhesions to all the surrounding tissues, so that it was quite impossible to consider any operation upon it. He accordingly performed a jejunostomy by the Witzel method. After the operation the patient vomited almost constantly. Very little food could be given by the tube and some of it returned through the wound. He rapidly lost weight and strength until he became a mere skeleton, and about three weeks after the operation died. An autopsy was performed by Dr. Damon R. Pfeiffer. His notes are as follows:

The body was much emaciated and the abdomen scaphoid. An incision was present in the upper right rectus muscle, about 12 cm. in length, which was healed except near the lower extremity, where the sinus created by a jejunostomy tube was still open. The body had been embalmed.

On section the thoracic viscera presented no abnormalities. On opening the abdomen, numerous dense adhesions were found binding the jejunum, pyloric portion of the stomach, and adjacent gastrocolic omentum to the abdominal wall. These were apparently inflammatory, the result of operation. Freeing the stomach, it was found in its usual position, high up beneath the left diaphragmatic arch, but extremely narrowed throughout its entirety, and very dense and firm. On removal and opening for examination it was found that the entire wall was diffusely thickened, varying from approximately 1 to 2 cm. in thickness. On the greater curvature, about 6 cm. from the pylorus, an ulcerated area, about 2.5 cm. in diameter, was present, with thickened precipitous edges. The mucosa throughout was loosely attached to the thickened wall and was thrown up in large rugæ. The thickening stopped abruptly at the pyloric ring. The pylorus itself was patulous but not distensible, except with difficulty. The peritoneal surface of the stomach was smooth, except where inflammatory adhesions had been present, and in the neighborhood of the ulcerated area, where a number of small nodules, mucoid in appearance, were attached to the stomach both on the greater and lesser curvatures. The omenta in this situation were thickened and contained a number of lymph nodes, varying in size up to 1 cm. in diameter.

The liver was atrophic but otherwise normal in appearance, except for cholecystic adhesions the bile passages were free and normal. The pancreas showed no abnormality, but in the peripancreatic and peri-aortic tissues of the neighborhood a few moderately enlarged lymph nodes were present.

The remaining abdominal viscera were normal in gross appearance.

Microscopically the edge of the ulcer showed on its gastric surface, an enormous hyperplasia of tubular glands, which correspond in appearance rather closely to the glandulæ gastricæ. They are, however, more closely set, without the usual intervening stroma and the lining cells are much larger and of the mucoid type. In some areas they are quite irregular and are undergoing mucoid degeneration *en masse*. In certain areas the appearance strongly suggests that of "Brunner's glands." That the invasive tendency is relatively slight is shown by the fact that this hyperplastic epithelial mass is quite well circumscribed from the underlying fibromuscular stomach wall, though in many places a distinct basement membrane is absent. In other places distinct evidence of invasion is seen.

The submucosa is not distinct from the muscularis with which it merges as a dense thickened fibromuscular layer. Here and there in the intermuscular spaces, apparently corresponding to the lymphatics are found strands and clumps of cells, evidently derived from the epithelium of the growth above described. There is no evidence that the ulceration was a primary condition with secondary

carcinomatous implantation, on the contrary the ulceration appears to result from degeneration of the hyperplastic epithelial mass. The invading cells within the fibromuscular layer occasionally reproduce a tubule; but for the most part they exist as simple strands without definite arrangement. They can be observed moreover in all stages of degeneration. Apparently, either by reason of compression by the surrounding tissue, or as a result of essential biological characteristics the individual cells show a marked tendency to undergo mucoid degeneration and necrosis. Where the cells are enclosed by dense fibromuscular tissue they tend to disappear completely, but where no surrounding pressure is exerted, as on the exterior of the stomach or in the lesser omental tissues as hereafter noted, their disappearance results in the deposit of considerable masses of mucoid material. That these masses are the result of degeneration of the cell bodies themselves can be inferred by the presence of cells in all stages of degeneration and fusion with the mucoid material.

Sections from the thickened gastric wall at various points, distal and proximal to the ulcerated area, show with only minor variations the same process. The obvious interpretation of this growth and the excessive thickening of the gastric wall is as follows: The greater curvature is the seat of the primary colloid or mucoid carcinoma which possesses invasive characteristics with low cellular vitality. Infiltration takes place along the tissue planes permeating the lymphatics of the entire stomach. As a result of degenerative processes the invading cells tend to undergo mucoid change and death. It seems possible that the presence of this cellular infiltrate and its products result in a hyperplasia of the muscular and fibrous tissue of the stomach, thus explaining the thickening of the wall. That a functional hypertrophy is not the cause of this thickening is shown by the fact that it is as great distal to the primary ulcerated area as proximal to it.

Section through the gastrohepatic omentum adjacent to the nodular area shows marked infiltration with a growth of the typical mucoid character. Sections from adjacent lymph nodes in the gastrohepatic and gastrocolic omenta also show in some instances infiltration with the typical cells of the growth. One small periaortic node from the region of the celiac plexus 0.5 cm. in diameter presents a similar infiltration. In the lymph nodes the globular cells lie closely in the marginal sinuses and central lymph spaces, and show the same degenerative tendencies but without the permanent deposit of mucoid material.

The pancreas and remaining intra-abdominal organs show no noteworthy abnormalities and no evidence of metastatic nodules. Noteworthy features are: (1) The suggestion of a derivation of the growth from Brunner's glands; (2) the evidence of diffuse infiltration of the stomach wall by the growth, with its pronounced

degenerative tendencies and the probable irritative hyperplasia of the fibromuscular coats; (3) the distinct evidence of extragastric lymphatic extension, which, however, in view of the duration of the condition, must be regarded as a late or exceedingly slow process.

The interesting features of this case appear to be as follows:

1. The development of symptoms one month after the operation for hernia raises the question whether the latter could have had anything to do with the subsequent gastric condition. If it had there is no possible way of demonstrating the fact. There was not at the time nor at the subsequent operation aside from the omental adhesions any evidence of infection of the hernial wounds.

2. The persistent patulous condition of the pylorus is unusual for, with one exception, in all cases hitherto recorded obstruction and retention of the gastric contents has been present.

3. The persistent absence of free hydrochloric acid, even though the total acidity sometimes reached 68, is somewhat difficult to explain.

4. The fact that pepsin was undoubtedly secreted, although, in the absence of free hydrochloric acid, it did not act upon proteids, but it always became active as soon as the gastric contents were diluted with hydrochloric acid solution. Pus was frequently found at first; occult or visible blood never; lactic acid never in this case, and although it has been observed in some of the recorded cases, it is not constant and probably only occurs when there is stagnation of the gastric contents.

5. The evidence at the time of operation that the process had begun in the pyloric region. The wall in this region was distinctly thicker, somewhat harder and appeared more altered than the remaining portion of the stomach, although the thickening of the gastric wall extended as far as the fundus.

6. The distinct evidence of improvement that occurred twice during the course of the disease, once when the patient gained ten pounds in weight and the other time when he became so much stronger that he was able to undertake a considerable degree of out door exercise walking as much as four or five miles across country almost daily. Why this improvement occurred in view of the progressive nature of the process it is difficult to understand.

The treatment of this condition must of necessity be purely surgical, and it seems in view of several cases that have been reported in which either temporary improvement or even apparently a cure occurred, that, in the absence of a positive Wassermann, operation should be undertaken as soon as the diagnosis has been made. If the process is carcinomatous it may in the early stage be possible at least to attempt a gastro-enterostomy. If it is a sclerotic inflammation secondary to an ulcer a gastro-enterostomy offers considerable hope for permanent relief, as in the cases of Souper and Riesman. If neither of these operations can be attempted it

is possible that a jejunostomy may result as successfully as did that of von Eiselsberg. Of course, if the process is merely a part of a multiple serositis nothing can be done, but the patient will probably not suffer seriously from the exploration.

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CLINICAL STUDY OF PAIN ARISING FROM DIAPHRAGMATIC PLEURISY AND SUBPHRENIC INFLAMMATION.[1]

By Joseph A. Capps, M.D.,
CHICAGO.

In 1911 we reported the results of some experiments on the occurrence and distribution of pain, where the pleural membranes were irritated during the process of removing fluid from the cavity.[2]

We found that the visceral pleura is not sensitive to pressure or scratching of a wire. We found that irritation of the parietal pleura nearly always produced pain, sharp and localized over the area irritated and never referred to distant parts, *e. g.*, the neck and abdomen.

We found that irritation of the diaphragmatic pleura over the outer margin set up pain in the hypochondrium and often in the abdominal wall down to as far as the navel or even to the groin, while over the central portion of the diaphragm it caused pain in the neck, particularly along the trapezius muscle ridge.

From these experiments it seemed justifiable to conclude that

[1] Read at the meeting of the Association of American Physicians at Washington, D. C., May, 1915.

[2] An Experimental Study of the Pain Sense in the Pleural Membranes, Arch. Int. Med., December, 1911, vol. viii, No. 6, p. 717.

the marginal part of the diaphragmatic pleura receives its sensory supply from the lower six intercostal nerves and that the referred pain is attributable to an irritation of the corresponding sensory segments of the spinal cord, which endow the abdominal wall with sensation.

Also that the central portion of the diaphragm receives it sensory nerves from the phrenic nerve, which carries afferent impulses to the cervical cord, and sets up reflex pain in the corresponding spinal segments, namely, the third and fourth cervical.

To supplement these experiments we have observed the character and location of pain in a series of clinical cases in which there was inflammation of the serous membranes lining the upper and lower surfaces of the diaphragm.

Analysis of Sixty-one Cases of Diaphragmatic Pleurisy. During a six-year period sixty-one cases of diaphragmatic pleurisy have been seen and examined by the writer. Such a collection would have been impossible had it not been for the interest and courtesy of the internes and staff of the Cook County Hospital.

Twenty-nine of the cases had a lower lobe pneumonia with associated pleurisy, the remaining thirty-two had pleurisy alone.

Thirty-nine were situated on the right side, twenty-one on the left and one was bilateral. The diagnosis was in most instances made evident by physical findings in the thorax, in four instances confirmed by autopsy, and in six instances abdominal disease was excluded by exploratory laparotomy.

Pain referred to parts distant from the seat of inflammation was the outstanding symptom of these cases. *Referred abdominal pain* was present in fifty-four cases, in all cases involving the upper quadrant (7 to 10 dorsal segments), and in twenty-five cases the lower quadrant as well (7 to 12 dorsal segments). Bilateral pain in the belly was noted four times.

The pain was usually spontaneous and with it was tenderness to pressure. *Hyperesthesia* and *hyperalgesia* of the skin were almost constant and showed a decided tendency to follow band-like areas, corresponding to different spinal segments. Over this sensitive area the *musculocutaneous* reflexes were exaggerated and often the wall remained in a state of rigidity.

The *maximum points of pain* and tenderness to pressure were especially studied. (Fig. 1.)

This point was located:

(*a*) In the gall-bladder region, 10 times.
(*b*) Lateral to and slightly above the navel, 15 times.
(*c*) Near McBurney's point, 2 times.
(*d*) Over the navel, 1 time.
(*e*) Midepigastrium, 1 time.
(*f*) Splenic region, 1 time.
(*g*) Lumbar region, 2 times.

Referred neck pain occurred in thirty-three cases, or in a little more than one-half of the total. The pain was located by the great majority of patients along the trapezius ridge, occasionally over the shoulder cap and in the supraclavicular space. These areas all are supplied with sensory nerves by the third and fourth cervical spinal segments.

The pain was usually spontaneous and of a sharp stabbing character, always well localized by the patient with the tip of his finger. Pressure over this point was often very painful. The skin and subcutaneous tissues in the immediate neighborhood showed the same hyperalgesia and hyperesthesia as were found over the abdomen, but the point of maximum pain and tenderness in the neck was sharply localized, and more constant.

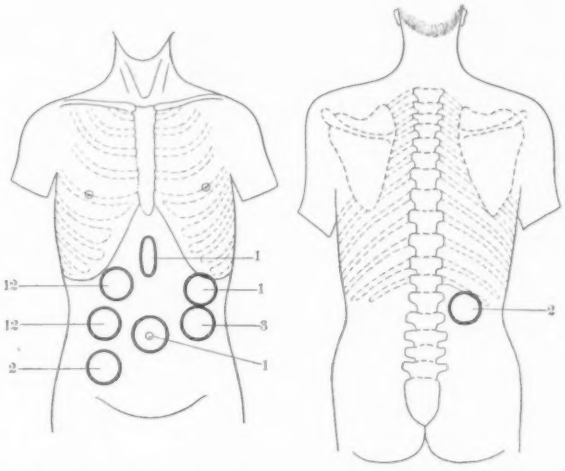

FIG. 1.—Points of maximum pain and tenderness in abdomen and back occurring in 61 cases of diaphragmatic pleurisy.

The exact locations of maximum pain points are illustrated in Fig. 2.

FACTORS INFLUENCING PAIN. The appearance of referred pains in the abdomen or neck was generally early in the course of the disease. More often abdominal pain preceded the neck pain when both were present. The *duration* of the pain and tenderness varied in much the same way as in parietal pleurisy. Sometimes the pain disappeared after a few hours; as a rule it persisted for one to three days and occasionally for over a week.

The pain was nearly always induced or aggravated by cough and deep inspiration, especially during the acute stage.

Measures limiting the excursion of the diaphragm on the affected side, such as lying on the painful side or tight pressure of an abdominal band often gave relief. Some patients secured ease by lying doubled up on the affected side with the shoulder held down by a pillow.

Special Features. The abdominal pain was associated with a maximum point of pain and tenderness far more frequently on the right than on the left side. Such a point was observed in 25 out of 36 cases on the right side or 70 per cent.; while it was noted in only 7 of 18 cases on the left side, or 40 per cent.

In contrast to this disparity, the neck pain occurred with almost equal frequency on the two sides. *Tenderness of the phrenic nerve,*

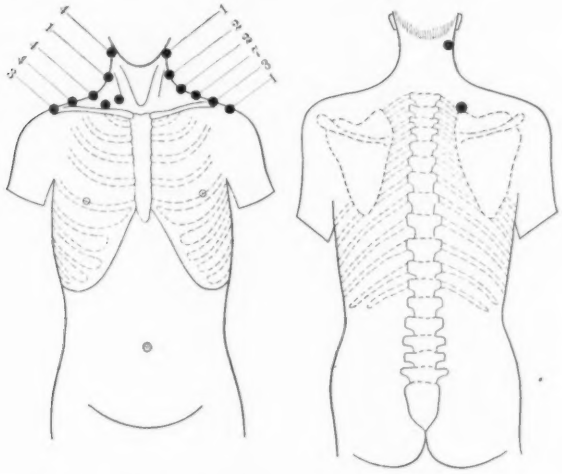

Fig. 2.—Points of maximum pain and tenderness in the neck region occurring in 61 cases of diaphragmatic pleurisy.

a symptom emphasized by many writers was found in but one case and that was doubtful. *Vomiting* was present in 10 cases and was often responsible for a mistaken diagnosis of abdominal inflammation. *Hiccough* another symptom supposed to be common in diaphragmatic pleurisy, was noted in only 5 cases, being unusually severe, however, in three.

Does the Referred Pain Originate in the Lung or Pleura? Head, Russell, and others have held that the irritation comes from the lungs, as they question the capability of the pleura to give rise to referred pain. In our series pneumonia of the lower lobe was present with pleurisy in twenty-nine cases. In thirty-two cases pleurisy was present without any signs of consolidation. This

indicates that the pleura over the diaphragm does cause referred pain and casts doubt upon the ability of the lung itself to do so. The pleural origin of referred pain is further corroborated by our experiments[3] in which irritation of the lung set up no pain, whereas, irritation of the diaphragmatic pleura gave rise to referred abdominal and neck pain.

Mistaken Diagnoses in Diaphragmatic Pleurisies. *Appendicitis* was incorrectly diagnosed in 9 cases. Two of these were operated upon and the abdominal viscera found to be normal.

Cholecystitis was diagnosed in 6 instances, 2 of which came to laporatomy without discovering any pathological condition of the gall-bladder or the neighboring structures.

Ulcer of the stomach with perforation was twice diagnosed and once operated upon without discovering any lesion in the belly.

Liver abscess was diagnosed in two patients. One was opened up with negative results. The other mistaken diagnoses included one instance each of *peritonitis, renal calculus, infectious lumbago,* and *brachial neuritis.*

Several of the above-mentioned patients were prepared for operation but were saved by the conservatism of the surgeon. I believe the unusual interest in referred abdominal pain awakened by frequent discussion of the subject by the Staff of the Cook County Hospital has saved from needless operation many cases that in former years would have been promptly submitted to exploration.

Differentiation of Pain in Diaphragmatic Pleurisy and in Inflammation of Abdominal Viscera. Our knowledge of visceral pain has been so enriched by physiological investigators, more particularly by Hilton, Ross, MacKenzie, Head, Meltzer, Lenander and Hertz, that we should interpret our clinical findings in the light of their results. Without discussing their work in detail, we may recall that Lenander and MacKenzie have maintained that the viscera are insensitive to pain, since during operations under cocaine the stomach, intestine, appendix, gall-bladder, and other organs may be cut, burned, and scratched without producing any painful sensation. All pain proceeding from abdominal inflammation they believe arises from irritation of or traction on the parietal peritoneum, which is supplied with spinal nerve fibers that are specially designed to carry pain stimuli to the cord and brain. The sympathetic nerve fibers supplying the viscera, they say, do not have the power to carry painful stimuli.

These conclusions must be modified by the observations of Kast and Meltzer, who found that the sensibility of the viscera during such operations is appreciably lessened both by the general toxic effect of cocaine and by exposure to the air.

It has been pointed out by numerous investigators that the

[3] Loc. cit.

viscera may be insensitive to the ordinary painful stimuli such as cutting and burning and yet be sensitive to the "adequate stimulus." The adequate stimulus for all the hollow viscera seems to be *tension*. As a result of tension the pain of colic may be explained.

The theory that generally prevails today is that proposed by Ross and supported in essentials by Head, viz., that there are two distinct types of pain in visceral inflammation.

1. *The splanchnic or visceral pain*, which is conveyed directly to the cord and brain by the splanchnic nerves. This pain is, as a rule, dull and deep. A good illustration of this pain is seen in pressure on the testicle or over an inflamed ovary, appendix, or gall-bladder. As a rule, it is localized near the site of its origin.

2. *Somatic or referred pain* is usually located at some distance from the seat of inflammation. It is produced when afferent stimuli from the viscera travel along the splanchnic nerves to the posterior roots of the cord, where they so irritate the corresponding spinal somatic nerves that they give rise to pain in the area of skin and muscles supplied by these nerves.

This pain has a band-like distribution over the trunk and an irregular distribution over the neck, head, and extremities. The pain is in the skin, subcutaneous tissues and muscles and therefore superficial. The sensitized area is hyperesthetic, hyperalgesic and often characterized by painful tender points. The muscular cutaneous reflexes are exaggerated. When the parietal peritoneum is involved then there is direct irritation of the spinal somatic nerve which is similar in kind to referred pain along the same nerves. It is reasonable to expect that both types of visceral and referred pain may be present in visceral inflammation. But clinical experience seems to show that the visceral pain is more constant in abdominal affections than the referred, also that visceral pain may occur alone, *e. g.*, in intestinal colic and some cases of catarrhal appendicitis. Referred pain from the diaphragmatic pleurisy renders painful the spinal nerves supplying the abdominal wall, but the deep visceral pain is not present. Steady deep pressure over the appendix or gall-bladder is well tolerated, whereas in inflammation of these organs deep pressure gives rise to severe deep-seated pain.

The main points of differentiation between diaphragmatic pleurisy and inflammation of the abdominal viscera are as follows:

1. The skin and muscles of the abdomen are more sensitive to pain and touch in referred pleural pain than in visceral disease. This is best elicited by pinching of the wall and scratching of the skin.

2. The cutaneous reflexes are more lively in referred pain than in visceral disease as a rule.

3. Deep pressure with the flat surface of the fingers is well borne in referred diaphragmatic pleural pain, while it elicits a dull, deep

pain when applied over an inflamed organ, *e. g.*, an appendix or gall-bladder.

4. Evidences of respiratory infection are usually present in diaphragmatic pleurisy, such as cough, expectoration, herpes of lips, sore throat, high leukocytosis, rapid respiration, etc.

5. Appearance of a sharp, definitely localized pain in the neck on the same side as the abdominal pain often reveals the true condition, since it points to irritation of the phrenic nerve.

6. The referred pains in the neck and abdomen are usually induced or aggravated by cough and deep inspiration.

7. Nausea and vomiting are more constant in visceral abdominal inflammation, but may occur and be very pronounced also in diaphragmatic pleurisy.

8. Hiccough is not a common symptom in diaphragmatic pleurisy, contrary to the current belief. It was present only five times in our series of sixty-one cases. It is more often seen in visceral diseases of the abdomen than in diaphragmatic pleurisy.

Subphrenic Inflammation. In the study of pain distribution from subphrenic inflammation, we are greatly handicapped. The opportunities for experimental irritation of the under surface of the diaphragm in human beings are rare. Moreover, the existence of abdominal pain in the course of subphrenic inflammation may be properly attributed to associated inflammation of the abdominal viscera. Only in those cases in which definite, sharply localized pain and tenderness develop in the neck or shoulder region, can we be sure that the diaphragm is involved. We have collected six cases of subphrenic inflammation exhibiting neck pain, three on the left side and three on the right. The painful point is in every respect identical with the referred pain in diaphragmatic pleurisy.

The nerve supply of the peritoneal covering of the diaphragm is probably the same as that of the pleural surface, that is to say, the lower intercostal nerves supply the outer margin, and the phrenic nerves supply the central portion. Hence it is impossible to differentiate between supraphrenic and subphrenic inflammation by the pain alone. The symptomatology and physical findings must also be considered, in order to establish the diagnosis.

THE CEREBRAL NERVE DISTURBANCES IN EXOPHTHALMIC GOITRE.

By George J. Heuer, M.D.,

(From the Department of Surgery, the Johns Hopkins University.)

The cerebral disturbances which occur in exophthalmic goitre are among the rarer manifestations of this remarkable disease. Headache, often intense, is a very common symptom, as are

irritability, emotional outbursts, and disturbances in sleep with terrifying dreams. Graver associated conditions may occur, such as neurasthenia, hysteria, and the various psychoses, such as manic-depressive insanity, melancholia, dementia precox, paranoia, and acute delirium. Organic cerebral changes giving rise to convulsions, epilepsy, and epileptiform attacks, muscular weakness, paralyses of the cerebral nerves, hemiplegias and paraplegias, anesthesias, paresthesias, and hyperesthesias may be associated with this disease.

The chief purpose of this communication is to call attention to one group of these cerebral disturbances, namely, the cerebral nerve palsies. In the enormous literature on Basedow's disease not more than eighty cases, including all types, are recorded, the great majority of which have been observed in European clinics. Only four cases appear in American literature (Campbell Posey, Dana, Gordon, Bartholow). At the Johns Hopkins Hospital, among more than 300 cases of exophthalmic goitre occurring in the service of Dr. Halsted, the case to be reported is, with the exception of an occasional mild grade of ptosis, the first instance which has come under observation.

The cerebral nerves affected are most commonly those controlling the movements of the eyes and lids, but a review of the literature shows that almost every cerebral nerve has been involved, either alone or in combination with other nerves. Least common is the group of cases with an associated bulbar paralysis, of which perhaps only ten have been reported. The case to be described is of this group.

The patient, a Russian Jew, aged twenty-three years, a recent graduate in medicine, was admitted to the surgical service of the Johns Hopkins Hospital January 31, 1911, complaining of weakness, vomiting, shortness of breath, difficulty in swallowing, and double vision. The family history is unimportant. The mother is in rather poor health and nervous. There is no history of exophthalmic goitre in other members of the family. The patient has always been delicate and of a nervous temperament, but has not had any severe illnesses. He was admitted to the medical service of the Johns Hopkins Hospital December 17, 1907, complaining of weakness and vomiting. The history which he gave at that time was as follows:

Two years before (in 1905) he had an attack of vomiting lasting for a few days; a year later he had a second similar attack; the third attack (for which he was admitted to the medical service) had been of two months' duration, and had increased in severity so that even small amounts of milk could not be retained. He had lost thirteen pounds in weight in the week immediately preceding his first admission. The vomiting had not been associated with nausea or pain; the food ingested was expelled without effort

or distress. During the few days before admission he had had diarrhea, four to five movements daily. Examination at that time (in 1907) showed an emotional, nervous young man with normal pulse and temperature; no exophthalmos or characteristic eye signs, apparently no enlargement of the thyroid gland, and no cerebral nerve disturbances. The lungs were clear; there was a faint systolic blow at the apex of the heart; the abdomen was negative. Acetone and diacetic acid were present in the urine. Dr. Barker noted, at this time, pigmentation of the skin and the absence of axillary hair. The patient remained in the hospital but two days, at the end of which time he insisted upon going home.

Present Illness (January 31, 1911). The patient states that he was perfectly well until March or April, 1909. He then noticed a swelling of his neck, which at first was not accompanied by any symptoms. Just previous to its appearance he had been roughly handled by his fellow-students and badly frightened. The enlargement of the neck was very rapid. Three months after the first appearance of the goitre he was unable to wear a collar, and had become very nervous. Tachycardia was first noted several months after the appearance of the goitre, and has persisted, but without subjective palpitation of the heart. A year after the appearance of the goitre (1910) he became aware of a droop of the right upper lid; there followed soon a similar condition of the left lid. This bilateral ptosis has gradually become more marked, and at present the patient is quite unable to raise the lids. Soon after the appearance of the ptosis he was troubled with double vision, a condition which has persisted. About five weeks before admission, owing to loss in the power of mastication, he was unable to chew solid food. In the past month he has had difficulty in speech; he has jumbled his words and states that he has "talked through his nose." A week before admission he completely lost his voice for a period of three days. For the same period his tongue felt thick; there has been a collection of mucus in his throat; he has had frequent cough and marked subjective dynpnea. There has been difficulty in swallowing, and fluids taken by the mouth have been repeatedly regurgitated through the nose. The patient has, however, been able to attend to his medical duties until three weeks ago, when great weakness of the upper and lower limbs compelled him to give up his work.

Examination. The striking feature on inspection was the patient's facial appearance. The drooping lids, the protruding, fixed eyes, the mask-like face, the open mouth and hanging jaw made a truly striking picture (see Fig. 1). He was weak, perspired profusely, and looked ill. He was unable to raise his head from the pillow, could scarcely raise his shoulders, and only with the greatest effort lift his arms from the bed. Dyspnea was so great that he was compelled to sit up in bed. Unable to expel the mucus

which collected in his throat, he had frequent, violent paroxysms of coughing. His voice had a distinctly nasal quality. He was clear mentally, but forgetful. He was emotional, and repeatedly burst into tears without assignable cause.

The exophthalmos was extreme. The other eye signs could not be tested on account of the complete bilateral ptosis and fixation of the globes. The thyroid gland was much enlarged, the right lobe more than the left, and was fairly soft and smooth. There was marked pulsation of the entire goitre, but no palpable thrill. On auscultation, a loud, systolic bruit could be heard over the entire gland; it was loudest over the superior thyroid vessels.

FIG. 1.—The striking facial appearance; the drooping lids, protruding fixed eyes, mask-like face, open mouth, and hanging jaw.

The pulse was regular and almost constantly about 120 per minute; the blood-pressure was 120 mm. Hg. The heart was not enlarged either to the right or left. A faint systolic bruit was heard over the apex; the sounds at the base were clear. There were no edemas. There was a well-marked tremor of the fingers. The hands and feet were sweating. Pigmentation was pronounced, particularly over the flexor and extensor surfaces of the extremities and about the trunk. Over the lower legs were several more or less circumscribed, brownish areas, and around the ankles there was quite a definite band of pigmentation. The skin of the abdomen was dark, especially about the waist line, where it was coffee-colored. There was some pigmentation of the skin of the forehead, deep

pigmentation about the genitalia, but none of the mucous membranes of the mouth.

Nausea, vomiting, and a rather persistent diarrhea, complained of during his illness, were absent during his stay in the hospital.

Blood Examination. Red cells, 5,600,000; hemoglobin, 100 per cent.; white cells, 9000; polymorphonuclears, 71 per cent.; eosinophiles, 1 per cent.; large mononuclears, 10 per cent.; small lymphocytes, 12 per cent.; large lymphocytes, 2 per cent.; transitionals, 2 per cent.

Examination of the lungs and abdomen was negative.

In the urine there was a trace of albumin in two of the four specimens examined and a well marked acetone reaction in three of them. Wassermann reaction was negative.

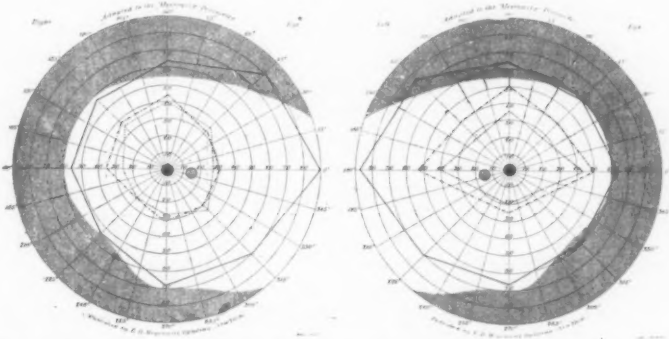

FIG. 2.—The eye fields, showing normal form fields, but contraction of the color fields, with interlacing of colors in right eye.

Cerebral Nerves. No disturbance in smell could be demonstrated. Taste also appeared to be fairly normal, although he miscalled salty and acid substances. There was complete bilateral ptosis of the eyelids. At rest the lids covered more than the upper half of either cornea. With the greatest effort the left upper lid could be raised slightly, but this movement seemed to be due rather to the elevation of the left eyebrow than to a true movement of the lid. The right upper lid could not be raised at all.

The greatly protruding eyes were practically fixed; there was no lateral, vertical, or rotary motion on either side. This total lack of movement of the globes persisted until the patient's death. Both eyes were directed forward in almost parallel lines. Double vision was constant, the upper image being the false one. Movements of the head from side to side had no influence on the position of the eyeballs, this test indicating an infranuclear rather than a supranuclear involvement. There was, therefore, a complete

ophthalmoplegia externa, a complete paralysis of the III, IV, and VI cerebral nerves. The pupils were usually but not constantly unequal in size, the left being larger than the right. They reacted promptly to light. The fundi were normal. The eye fields (see Fig. 2), showed normal form fields, but contraction of the color fields with interlacing of colors in the right eye.

The sensory fifth nerve appeared to be unaffected. There was no anesthesia of the conjunctiva and no sensory disturbance in the area of distribution of the trigeminal on either side of the face. The motor fifth, however, seemed markedly involved. The open mouth and hanging jaw were apparently due to the weakness of the muscles supplied by this nerve. At times the patient was unable to close the mouth; at other times he could, with a great effort, bring the teeth together. There was no evidence of contraction or atrophy of the masseters.

Nerve VII. Facial weakness was manifest on both sides, being more marked on the right than on the left. The face appeared placid and mask-like. The ordinary expressional movements were feebly executed. The patient could at times pucker his lips slightly and draw down the angle of the mouth; he was able to elevate the left eyebrow but not the right.

No disturbances in hearing could be demonstrated by the ordinary tests.

The voice, thick and indistinct, had decidedly a nasal quality; there was difficulty in articulation but no aphasia. The patient stated that his tongue felt stiff and awkward. Swallowing was difficult, and fluids taken by the mouth were in part regurgitated through the nose. There was a constant collection of mucus in the throat, which the patient seemed unable to expel. Cough was frequent and violent. Dyspnea was marked. The patient was able to protrude his tongue but slightly and the lingual movements were awkwardly performed. There was a marked tremor of the tongue. Examination of the palate and the pharynx was difficult. The palate appeared symmetrical. The pharyngeal muscles could be seen to move, but the examination was unsatisfactory. Sensation of the pharynx appeared to be normal. Examination of the larynx was unsatisfactory because of the patient's restlessness and the presence of mucus in the throat. Dr. Crowe stated the cords moved well on phonation.

The trapezius muscles were weak, the patient being hardly able to elevate his shoulders. Atrophy of these muscles was not demonstrable.

Paracentral Region. There were no gross sensory disturbances on either side of the body. There was, however, marked hyperesthesia of the skin. The patient complained whenever the skin was touched. This sensitiveness seemed especially pronounced over the abdomen and legs. The soles of the feet were extremely

sensitive, and the patient objected bitterly whenever plantar stimulation was attempted. There was no definite paralysis of the extremities, but a profound, general weakness. As has been stated, the patient was unable to support his head or to elevate his shoulders, he was barely able to raise his arms from the bed. The grip in either hand was practically *nil*. The muscular power in the legs was distinctly greater than in the arms. The muscular weakness seemed greater upon the right than upon the left side.

The reflexes were active at the knees and ankles. A suggestive ankle-clonus was obtained on one occasion. Plantar stimulation was unsatisfactory because of the extreme sensitiveness of the soles of the feet. The abdominal and cremasteric reflexes were normal. To Oppenheim's test there was a normal response.

Summary of Neurological Findings. Complete bilateral ptosis, complete ophthalmoplegia externa, paralysis or at least marked paresis of the motor fifth and of the seventh, bulbar paralysis, hyperesthesia of the skin, and profound muscular weakness.

Symptoms Referable to Involvement of Ductless Glands Other Than the Thyroid. The great muscular weakness, the pigmentation of the skin, and diarrhea suggested possible involvement of the *thymus* and *adrenal glands*. The pigmentation was most marked in places of pressure, that is, along the waist line, about the shoetops, and around the neck.

Symptoms suggestive of involvement of the *hypophysis* were also present. The body was quite fat, with a thick panniculus. The skin was soft, smooth, and feminine in appearance. The hips were rather broad. There was almost complete absence of axillary hair; the beard was less abundant than normal and there was absence of hair over the forearms and backs of the hands. The genitalia appeared to be normal. During the two months previous to admission there had been complete absence of all sexual desire.

Given the neurological findings which have been described, the question arose, Were we perhaps dealing with an organic lesion or lesions in the brain or with a myasthenia gravis? The picture presented by the patient corresponded closely with myasthenia gravis, which indeed has been observed associated with exophthalmic goitre. In that disease there may be ptosis, involvement of the eye muscles, the muscles of the face, of mastication, etc. All the voluntary muscles may become involved. Myasthenia gravis has been fatal in about 40 per cent. of the cases. Examination of the nervous system in this disease has revealed no abnormality. Of value in differential diagnosis is the myasthenic reaction of Jolly, the rapid exhaustion of the muscles by faradism. Our patient was tested for the myasthenic reaction on three successive days. Faradic stimulation sufficient to produce a good contraction of the muscles was employed. The muscles tested were usually the facial, the deltoid, and the quadriceps extensor of the leg. The stimuli were

applied at the rate of about 1 per second, and in all cases at least one hundred times, and sometimes for from two to five minutes. In none of the examinations could any fatigue of the muscles be demonstrated even after repeated stimuli. If this test is of value our patient was not a case of myasthenia gravis.

The patient remained in the hospital for six days. He refused operation. He was visited at his home, and in the ten days which elapsed between his first and second admissions there was no change in his condition. He finally consented to an operation, which was performed February 16, 1911, by Dr. Halsted. The condition of the patient was so serious that ligation of the superior thyroid arteries alone was done. Anesthetization was begun with the patient in a sitting position because of the orthopnea. His pulse rose to 170, and at one time to 180 during the operation, but at the end of the procedure his condition was satisfactory.

Postoperative Course. The patient was again carefully examined between eight and ten hours after operation. There was subjective improvement, the patient stating that he felt better than before the operation. Some slight movement of the globes was demonstrable, but this was not striking. His pulse was about 140. His temperature had risen to 101°. There had been some nausea following operation, but very little vomiting. He passed a fair night. During the following day (February 17) his pulse remained about the same, although at times it was slightly irregular. His temperature remained elevated. His blood-pressure, about 120, was unaffected. He eagerly took water, but the greater part of it was promptly expelled through the nose. There was a constant collection of mucus in the throat and frequent choking spells. About 8 P.M. of this day he complained greatly of shortness of breath, and became choked with mucus which he was unable to expel. His respirations suddenly ceased at 10 P.M., the heart continuing to beat. With the idea that there might be some obstruction to respiration a tracheotomy was promptly performed and artificial respiration undertaken. The heart continued to beat for perhaps five minutes during this procedure, and then ceased. Death, therefore, seemed to be due to an acute respiratory paralysis. Certainly, it was not a cardiac death, as is so often the case in exophthalmic goitre.

Most unfortunately an autopsy could not be obtained. A fragment of the thyroid gland, however, was removed, which showed the histological picture of Basedow's disease.

LITERATURE. The literature on cerebral nerve disturbances in exophthalmic goitre up to and including the year 1911 is considered with remarkable comprehensiveness by Sattler and Kappis. From these two authors chiefly and from the literature since 1911, I have obtained the following data as to the frequency of involvement of the various cerebral nerves.

I. Isolated palsies of the first or olfactory nerve have not been

reported in the literature, nor have isolated disturbances in taste. In combination with other cerebral nerve disturbances, loss of smell and taste has been observed in two cases of exophthalmic goitre (the cases of Warner and Bristow and of Ballet).

II. Isolated loss of vision has not been reported in exophthalmic goitre. Combined with other cerebral nerve disturbances the optic nerve has been affected in one case (Ballet), which showed diminished visual acuity on the left side with contraction of the visual field.

III. The third or oculomotor nerve has been most frequently affected and the majority of the cases reported have shown involvement of this nerve. Single muscles or all the muscles supplied by this nerve have been involved. Levator palsies and ptosis, either unilateral or bilateral, have been observed as isolated findings 9 times. Isolated rectus superior palsies have been reported 6 times and isolated rectus internus or convergence palsies 7 times. Of the latter, some observations seem questionable. Combined with other cranial-nerve disturbances the III nerve has been frequently affected. Isolated ophthalmoplegia externa, that is to say, palsy of the III, IV, and VI nerves, has been reported 6 times and an ophthalmoplegia externa combined with bulbar paralysis, as in our case, has been observed 5 times. The nerves supplying the internal muscles of the eye, *i. e.*, the pupils, have been affected in 5 instances.

IV. Isolated palsy of the fourth or trochlear nerve has been reported in one case; combined with other nerve lesions in 13 or 14 cases.

V. Isolated palsy of the motor fifth has not been observed; combined, this nerve has been affected 5 times, chiefly in cases of bulbar palsy. Involvement of the sensory fifth has not been reported except associated with a hemianesthesia.

VI. The VI nerve has, next to the III, been most frequently affected. Isolated palsy of this nerve has been observed 10 times. It has been frequently involved associated with the palsies of the III and IV nerves in opthalmoplegia externa and in the cases with bulbar paralysis.

VII. Isolated palsies of the VII or facial nerve have occurred 5 times; combined with other cranial nerve disturbances 8 times.

VIII. Isolated involvement of the auditory nerve has not been reported. Combined with other disturbances loss of hearing has been noted in one instance.

IX. Isolated palsy of the glossopharyngeal nerve has been observed once. Combined, it has been involved in association with other nerves in the cases with bulbar paralysis.

X. The pneumogastric has not been affected alone. Vagus disturbances, however, have occurred in the cases with bulbar paralysis.

XI. The spinal accessory appears to be the only cranial to have escaped involvement in exophthalmic goitre. No true palsy of this nerve has been recorded either isolated or combined. Marked weakness of the sternomastoid and trapezius muscle has been observed in association with general muscular weakness, as in our case.

XII. Isolated palsy of the XII nerve has not been reported. Combined, this nerve has been involved in the cases with bulbar paralysis.

It may be seen from this brief summary that the nerves controlling the eye muscles are most frequently involved in exophthalmic goitre. Kappis was able to collect over 40 cases in which the eye muscles alone were affected. Isolated palsies may occur, but combinations of various kinds are most commonly seen. A pure ophthalmoplegia externa, as previously stated, has been observed in 6 cases.

With the exception of palsies of the facial, 5 cases of which have been reported, isolated palsies of the remaining cerebral nerves are extremely rare. Combined palsies of these nerves, as recited, are not uncommon, and the most varied clinical pictures occur.

The cases with bulbar paralysis form a most interesting group, and deserve perhaps more than passing mention. They have all been severe cases of exophthalmic goitre, and in most instances the disease has run a rapid course. In one instance the patient had had a goitre for many years, on which Basedow had become engrafted.

The duration of the disease has varied from a few days to five or six months before the onset of bulbar symptoms. Death has invariably followed the appearance of these symptoms, and in the majority of instances within a short time. One case died one day, another three days, and another eight days after the onset of bulbar symptoms, while others have lived from several weeks to three months thereafter. We may, therefore, divide the cases into those with acute (the majority) and those with chronic bulbar palsy. Our case is of the latter group. While there has been considerable variety of combination in the nerve palsies, the bulbar symptoms have been remarkably uniform in all the cases.

It is difficult, from the reports of cases in the literature, to differentiate the cases of bulbar paralysis due to organic lesion from those of myasthenia gravis with bulbar symptoms; and it is possible that some of the 12 cases assigned to true bulbar palsy do not correctly belong in that group. The cases were all reported between 1886 and 1904, and 8 of them before 1900. The myasthenic reaction was tested, apparently, in very few instances, if at all. We have, therefore, to base our opinion largely on the clinical course of the paralysis. Autopsy findings are suggestive but not altogether convincing. In 6 of the 12 cases an autopsy was not done, or no mention is made of it. In 4 of the remaining 6 cases a definite

lesion was found in the nuclei of the affected nerves, described either as an area of softening or of fresh encephalitis. In the remaining 2 cases it is stated that gross lesions of the brain were not present; but serial sections for microscopic examination were not made, and therefore these negative findings are inconclusive; 2 of the 12 cases certainly suggest a myasthenia gravis chiefly because of the variability in the palsies. In neither of these 2 cases was an autopsy obtained.

ETIOLOGY OF NERVE PALSIES IN EXOPHTHALMIC GOITRE. As regards the etiology of nerve palsies in exophthalmic goitre, it may be said that no very definite knowledge is at hand. It is assumed that the disturbances in the cerebral nerves are of a toxic nature, the toxins being responsible for the lesions in the nuclei of the cerebral nerves. Clinically, there is some evidence for this assumption, for palsies occur, as a rule, after the disease is well established, and never, so far as is known, in cases of cured exophthalmic goitre. Cases are known, however, though they are very rare, in which palsies appeared shortly before the outspoken symptoms of Basedow's disease, while in a few instances the palsies and the usual symptoms of exophthalmic goitre have appeared simultaneously.

PATHOLOGY. In 4 of the 6 cases with bulbar paralysis in which autopsies were obtained, definite lesions were present in the medulla associated with extensive degeneration of fiber tracts. The 2 cases with negative findings are not conclusive, for a careful examination of the brain was not made. It has been in the cases with bulbar paralysis that the most positive pathological findings have been obtained, for they have all been severe cases of exophthalmic goitre which have quickly terminated fatally. In the great majority, the palsies have affected chiefly the eye muscles and apparently have not influenced greatly the course of the disease. These cases have gone from observation and the end result has not been known. In brief, it may be said that comparatively few pathological lesions in the brain have been recorded in the cases of exophthalmic goitre with nerve palsies. This may perhaps be explained (1) by the fact that they rarely have been looked for, (2) because the majority of the patients have not died while under observation, and (3) because serial sections of the brain have not been made—a necessity for the demonstration of a small lesion.

THE CLINICAL COURSE OF THE CEREBRAL NERVE PALSIES. The palsies may appear at any stage of the disease. In most cases they manifest themselves months or even years after the onset of the disease; in one case it was not until six years after the first symptoms of exophthalmic goitre that the palsies were noted. They may, however, begin acutely, simultaneously with or soon after the other symptoms. In a few instances the palsies have been reported as the first symptom of Basedow's disease. In most cases they

have entered the clinical picture almost unobserved, the disease not having become noticeably more serious with their occurrence. But the palsies may, as stated above, begin acutely and spread rapidly. In some of the cases reported it has developed over night, and synchronous with the rapidly spreading palsies there has been a marked increase in the severity of the disease.

In cases in which several nerves were involved there seems to have been no uniformity as to the order in which the paralysis appeared. Frequently they have occurred almost simultaneously, but they may follow one another at irregular intervals. The period between the onset of the first palsy and that of the last has not been great—less than a year in the vast majority of cases, but two years in a patient of Kappis. The complete paralyses have usually persisted unchanged. In no case has the palsy been benefited by operation, although marked improvement in other symptoms has been noted. There are apparently 2 cases, however, in which palsies of short duration have disappeared on improvement of the other symptoms, and 3 cases in which there was a partial recovery of the palsies.

What has been said thus far regarding the clinical course of these palsies does not hold for the cases with bulbar paralysis. These cases have, without exception, been highly toxic, the palsies have been rapidly progressive, and a fatal termination has quickly followed.

Differential Diagnosis. I shall merely mention some of the conditions in exophthalmic goitre which may simulate the true palsies. As has been said, myasthenia gravis undoubtedly may be associated with this disease. Sattler gives abstracts of 6 cases in which a positive myasthenic reaction was obtained. In addition to the myasthenic reaction a fairly characteristic finding in the cases of myasthenia is the variation from day to day in the palsies. Brain tumor has been noted in one case of exophthalmic goitre with nerve palsiess. Cerebral hemorrhage, multiple neuritis, and multiple sclerosis have all been observed associated with exophthalmic goitre, and it is possible that these complications might give rise to some difficulty in diagnosis.

Prognosis. A few additional words may be said as to the prognosis in the cases of Basedow with nerve palsies. Slight palsies, as those of the eye muscles, such as occur in the majority of the cases do not necessarily make the prognosis more grave. The paralyses themselves do not lead to death; the prognosis depends, as I have said, upon the severity of the Basedow. If the paralyses develop slowly, or acutely if merely to a slight extent, they have a serious significance *per se* only when they involve important nerves such as the vagus, which occurred in one case of Kappis. In the cases with very acutely developing and at the same time extensive paralyses the development of a bulbar paralysis is to be feared.

The occurrence of a bulbar paralysis makes the prognosis very grave, for, as has been previously stated, death has invariably followed.

TREATMENT. Only 4 cases have been operated upon. In 2 cases reported by Kappis the operation had no effect on the eye palsies, although the cardinal and other symptoms of exophthalmic goitre were greatly improved. So far as I am aware the case which I am reporting is the first of the grave ones (with bulbar paralysis) to be operated upon. Kappis reported a third case, one with difficulty in swallowing, which was believed to be due to paralysis of the glossopharyngeus and vagus. The patient died four hours after a lobectomy.

The presence of nerve palsies, excluding possibly the cases with bulbar paralysis, does not contra-indicate surgical intervention. Indeed, it would seem advisable to operate promptly in the hope not only of arresting the progress of the palsy but also of curing the disease.

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THE METABOLISM AND TREATMENT OF RHEUMATOID ARTHRITIS. THIRD PAPER.[1]

BY RALPH PEMBERTON, M.S., M.D.,

PHYSICIAN TO THE PRESBYTERIAN HOSPITAL, PHILADELPHIA.

(From the Department of Clinical Chemistry in the Pathological Laboratory of the Presbyterian Hospital.)

IN two previous communications[2] the writer has presented a series of cases of rheumatoid arthritis, 18 in all, in which certain laboratory findings and clinical observations were made the basis

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[2] The Metabolism and Successful Treatment of Chronic Joint Disease; A Preliminary Report, AMER. JOUR. MED. SCI., October, 1912, No. 4, cxliv, 474; and The Metabolism, Prevention, and Successful Treatment of Rheumatoid Arthritis; Second Contribution, ibid., December, 1913, No. 6, cxlvi, 895; January, 1914, No. 1, cxlvii, 111; February, No. 2, p. 265; March, No. 3, p. 423.

of a method of treatment. The results of this were sufficiently encouraging to justify and require a more extended study, with the idea of determining not only the proportion and kinds of cases in which benefit could be expected, but also, if possible, the *modus operandi* of the factors concerned.

The methods in question consist essentially in a curtailment of diet, particularly the carbohydrates and proteins, to a point of tolerance which varies with the individual.

An additional series of cases of rheumatoid arthritis, 19 in number, has accordingly been treated and studied. It is the purpose of this article, therefore, (1) to review the end-results of the first series, where this is possible, some of them dating back five years; (2) to present the results of treatment of the second series of cases, together with some analysis of the failures and successes; (3) to present the results of certain feeding experiments in which the effort was made to determine the specific action, if any, of some foods; and (4) to present the results of laboratory observations, especially upon the hydrogen ion concentration of the urine and the carbon dioxide tension of the alveolar air during the active stages of the disease and during convalescence or recovery therefrom.

End-results of the First Series. The first series consisted of 18 cases, and a review of them at the present writing brings out several interesting points. No attempt will be made to go into the details of the cases, as these have already been fully given.

Case I was first seen in January, 1911, and remains well, nearly five years afterward.

Cases II, IV, IX, XIII, XVI, and XVII have disappeared from observation.

Cases III and VIII were temperamentally unable, except under close observation, to adhere to any careful régime involving self-denial, and after becoming practically free from active symptoms, deliberately ate what they chose and relapsed.

Cases V and VI are entirely well and leading active lives.

Case VII has run a course of much interest. After making a symptomatic recovery, she slowly lost ground, and in July, 1914, a strict diet modified only slightly an active arthritis. In August she developed acute intestinal obstruction, was operated upon by Dr. John Kennedy, of Philadelphia, and a stenotic band was relieved, after which her arthritis was greatly improved. She felt so well that she ate much more generously, adhering only slightly to the lines of her earlier diet. Her arthritis grew slowly worse, and when last seen, March, 1915, she had definite symptoms, though less than at some previous periods.

The beneficial influence of diet in this case when first treated, the violent exacerbation just preceding acute intestinal obstruction, the relief of symptoms during a long period following the operation, and the gradual increase of the arthritis at first influenced

by a diet and then increasingly less so form a most suggestive sequence of events.

Case X remains well, on duty as a hospital attendant.

Case XI, who was bed-ridden, is free from active arthritis, gets dressed, and walks around one floor of her home. Her chief limitation comes from the ankylosis of her knees in extension, present when first seen. It is of interest to note that there has been present throughout an active leucorrhea which was not properly treated because of the inability to abduct the patient's thighs. This still persists, though the thighs can now be abducted. Her original and subsequently maintained improvement in the face of an obvious source of possible infection is of much interest.

Case XII is reported as active and walking without crutches, and Case XIV is on active duty as a nurse.

Case XV is in buoyant health, and has acquired a considerably increased tolerance for food.

The degree to which patients in this series have differed in their caloric necessities has been noteworthy. Several of them have maintained constant weights upon fewer calories than are generally regarded as practicable in ambulatory cases, as previously noted.

New Series. Space will not permit of giving more than a bare outline of the cases and treatment in this series, together with certain limitations to treatment, except where some particular point necessitates it. In the previous series the full details of each case, together with frequent notes showing the changes of diet and progress, make a repetition of these data in connection with the present cases unnecessary. It should be stated that every case but one was Roentgen-rayed by Dr. W. S. Newcomet, director of the Roentgen-ray department of the Presbyterian hospital, and the clinical diagnosis of arthritis was always thus corroborated. Nearly all of the chronic cases had been through more or less treatment, and some had experienced a great variety.

Case XIX.—Mrs. L., aged sixty-four years, referred by the courtesy of Dr. Walter L. Mendelson, of New York City, was admitted March 28, and discharged June 29, 1914. She presented exophthalmos, enlargement of the thyroid, some tachycardia dating back twenty-five years, an enlarged heart and murmurs. The arthritis was of ten years' duration, affecting the knees, shoulders, and hands. For ten days the patient was watched upon an unrestricted house diet and lost three pounds in weight, averaging 2346 calories per diem, and on April 6 she was given a mixed diet of 1385 calories. By a mixed diet is meant one consisting roughly of one or two eggs, from two to six 30 gm. slices of bread, about 50 gms. of chicken, 15 to 30 gms. of butter, and about 15 gms. of sugar plus green vegetables, according to the desired total. In three days there was some improvement, and on the eleventh day there was no pain in any finger or in the wrist on pressure.

June 17 ,1915. Heas ben doing progressively well for two months. For a month the effort has been made to increase diet, body fat, and weight. Today she feels for the first time some tenderness in the last joint of right ring finger.

The patient left the hospital June 29, with greatly increased bodily activity and hands free except as per last note. Weight, 123 pounds; loss, 16 pounds. She spent the summer at a hotel and followed the diet as well as she could, but grew restive under it. She was seen with Dr. Mendelson in November, 1914. The arthritis of the right ring finger was worse and one other small joint was involved. Her bodily activity, knees, and shoulders were much improved, especially the former two.

Retrospect of this case reveals that the hyperthyroidism was a complicating factor and added to the difficulties of treatment, in that the patient ran an increased pulse-rate throughout and tended easily to lose weight. The long stay in the hospital helped her tremor but made her restive. Her diet was increased too soon, and she admitted that after leaving the hospital she had taken some liberties with it. The entirely favorable application of diet to her arthritis was beyond question, however. The result from her stand-point was an improvement, modified and limited by the complicating hyperthyroidism and by the personal element which led to a curtailment of treatment short of the theoretical ideal.

Case XX.—Mr. F., aged about fifty-five years, was referred by the courtesy of Dr. Milton J. Lichty, Cleveland, Ohio. Admitted April 9, 1914, discharged July 6, 1914. An arthritis of four and a half years' standing involved practically all joints, and he was confined to a wheel-chair. A probationary house diet showed 3500 to 4000 calories. After several changes the new mixed diet was maintained at 1185 calories. Improvement in the hands began in six days. No changes occurred in the ankylosed joints, but when the patient left the hospital on July 6, 1914, his hands had greatly changed their appearance and he had less pain generally. The loss of weight in three months was twenty-four pounds. This patient was beyond everything but the arrest of the disease in its then stage, but there cannot be any doubt that the causative arthritis was emphatically modified, and in the smaller joints had largely subsided when he left.

Case XXI.—Mrs. H., aged sixty-three years, was referred by the courtesy of Dr. Hobart A. Hare. She presented an arthritis of four years' duration, involving the knees, shoulders, and hands She was obese, with myocarditis and loud murmurs. A probationary house diet averaged 2024 calories. She was given a mixed diet of 1271 calories and after six days showed a beginning subsidence of inflammation in the hands. She developed cardiac irregularity and beginning loss of compensation after two weeks of restricted diet, requiring complete rest in bed and stimulation. This diet

was insufficient to accomplish permanent results, and evidently a further reduction was necessary. In view of the heart condition, however, such a step was regarded as dangerous. The patient adhered faithfully to the above diet for three months without any additional improvement in the hope that, though insufficient to arrest the causative arthritis, something might be accomplished. The end-result to her, however, was absolutely *nil*. This case is of value as illustrating the fact that in face of a beginning and distinct response to treatment, as evidenced in the hands, limitations from another source may preclude further efforts. It is important on many counts, as illustrated above, to keep the caloric total as high as possible, from which it follows that in many cases several or more diets have been required before the proper reduction was obtained.

Case XXII.—Mr. P., aged thirty-one years, was referred by the courtesy of Dr. Penn G. Skillern. An arthritis of nine months' duration, immediately following a severe infection of forearm, incapicitated him for work by involvement of hands, knees, and shoulders chiefly. There was no apparent focus of infection anywhere. The patient was a large eater, and began to recover upon a mixed diet of 2500 calories, which were reduced in ten days to about 2000. Progress was steady from the start, and he went home well. Admitted March 3, 1914. Discharged May 2, 1914. Last heard from and well October 15, 1915. The loss of weight in hospital was from 134 to 127 pounds.

Case XXIII.—George W., aged forty-five years, ward case, was admitted June 26, 1913. An arthritis of eight months' duration, involving knees, heels, shoulders, and hands, incapacitated him. His condition was febrile and somewhat suggestive of acute inflammatory rheumatism superimposed on the chronic form. He was given a very low mixed diet of 629 calories for ten days. Undoubted subsidence of the acute symptoms followed. The calories were increased to 1072. The case was not followed beyond July 12, 1913, but the evidence was definitely favorable until then.

Case XXIV.—Mrs. McC., aged fifty-four years, admitted May 25, 1914, discharged August 20, 1914, presented a chronic arthritis of uncertain duration. There were also redness, tenderness, and effusion in knees, ankles, hands, and wrists. Practically every joint was involved, and the symptoms pointed to an acute and severe inflammatory process on top of the chronic form. She was given five glasses of milk in twenty-four hours, increased by eggs chiefly, in course of one week to 1011 calories. Her joints responded from the start, becoming in six days practically painless. Within two days after admission she developed signs of pneumonia, and had a delayed recovery therefrom. The calories were later increased to 1950 to meet the convalescent demands. In January, 1915, her general condition was excellent and the joints were in tolerably

good shape. The social condition of the patient precluded intelligent coöperation.

Case XXV.—Mrs. R., aged thirty-three years. Was admitted May 22, 1914, discharged July 10, 1914, and had an incapacitating arthritis of three months' duration involving hands, elbows, and knees. Bony deformity of the fingers was present. A probationary house diet averaged 1916 calories. On a mixed diet of 1314 calories she improved at once and steadily. She left the hospital almost free from all symptoms, and has remained entirely well since.

Case XXVI.—Mrs. S., aged forty-five years, was admitted May 13, 1914, and discharged May 22, 1914. An arthritis of three years' duration involved hands, wrists, elbows, shoulders, knees, and ankles. On a probationary house diet of 1734 calories she began to improve at once and left the hospital against advice when the subjective symptoms had subsided. This case is reported to illustrate the occasional improvement which follows upon the restricted régime of a hospital diet. Comment on this will be made later. Had she stayed a permanent limit of intake would have been assigned her, in the attempt to insure against relapse.

Case XXVII.—Mrs. C., aged thirty-five years, referred by the courtesy of Dr. A. A. Eshner, was admitted April 27, 1914, and left May 24, 1914. An arthritis of three years' duration involved both hands, wrists, elbows, shoulders, knees, ankles, and feet. A probationary house diet averaged 1933 calories. She improved on 1179 calories of mixed diet, but after ten days became dissatisfied with restrictions and left against advice.

Case XXVIII.—Mrs. F., aged thirty-nine years, was admitted February 8, 1915, and left February 27, 1915. An arthritis of two years' duration involved spine, jaws, shoulders, elbows, hands, and knees. Four months pregnant. Many fibrous nodules presented along ulnæ, tibiæ, and around patellæ. She showed some improvement on probationary house diet of 1494 calories, and was given 1396 calories of a fixed diet to insure constancy of intake. Rapid and unmistakable progress followed. She became dissatisfied with restriction of diet, and left against advice. The fibrous nodules had markedly subsided.

Case XXIX.—Mrs. L., aged about forty-two years, wife of a physician, was referred by the courtesy of Dr. D. J. McCarthy. She was admitted April 19, 1915, and discharged July 5, 1915. An arthritis of nine years' duration involved the shoulders, elbows, wrists, hands, and feet. There were fibroid nodules on both Achilles tendons. A probationary house diet yielded 1850 calories, and she was given a mixed diet of 1191 calories, reduced in two weeks to 1028. Progress was slow but sure. The fibrous nodules and three-quarters of the painful sites were practically gone when she left the hospital. She was instructed to follow her diet all summer

and report in the fall. The loss of weight was from 116 to 105 pounds.

Case XXX.—Mrs. W., aged about forty-nine years, wife of a physician, was referred by the courtesy of Dr. B. W. Sherwood, of Syracuse, N. Y. Admitted April 20, 1915, discharged June 12, 1915. An arthritis of two and a half years' constant duration, dating back irregularly thirteen years, involved the shoulders, elbows, and hands. This case was of much interest, and was a complete failure. The probationary house diet yielded 2048 calories. New diets were tried of 1294, 1159 and for a short time 674 calories, without clear improvement. An abdominal *x*-ray was not taken, as customarily on admission, as patient had had this done and the results were reported negative. Upon failure of treatment an *x*-ray was taken and showed a high degree of visceral stasis, partial collapse of the large bowel, evidence from the bismuth of obstruction in or near the ileum, chronic appendicitis, and possibly duodenal ulcer. The reason for failure cannot be advanced unless it be dependent upon these findings, especially the first two. In discussing the failure with Dr. Sherwood, before the *x*-rays were taken, the writer cited Case VII (*q. v.*), as supplying a possible explanation in the obstructive factors there operative. The patient's condition did not suggest, *a priori*, any difficulty of treatment.

Case XXXI.—Mrs. W., aged about forty years, was referred by the courtesy of Dr. John H. Girvin. A multiple arthritis had for some years involved shoulders, elbows, knees, and hands. Some improvement followed a probationary house diet of 1544 calories. The patient had bad tonsils, and these were removed. It is interesting to note that upon the two days immediately following the operation the patient felt great relief in her joints everywhere, during which time she was nearly without food. As the food was increased the pain and disability returned. The patient then went home to ascertain the end-result of the tonsillectomy, but of her own accord attempted to follow a diet of 1433 calories which she had been given for a few days at the hospital prior to the operation. At this point she disappeared from observation, but was reported later as doing very badly. The points to be noted in this case are the prompt and marked relief coincident with postoperative starvation, ceasing as ingestion of food increased; and the difficulty to be met when patients attempt dietary control by themselves before they are thoroughly accustomed by experience accurately to prepare and follow a diet. Earlier observations on the postoperative relief above recorded helped the writer to formulate the hypothesis upon which this study was started.

Case XXXII.—Mrs. W., aged about sixty-two years, was referred by the courtesy of Dr. Agnes Vietor, of Boston. Admitted March 9, 1915, discharged June 9, 1915. An arthritis of thirty

years' standing involved nearly every joint in the body except spine and jaws. Myocarditis and mitral regurgitation were present. A probationary house diet yielded 1945 calories. She was placed on a mixed diet of 1080 calories, and improvement began at once, especially in the hands. After one month the case "hung fire." The calories were changed to 1283, of which 753 came from fat. The joints improved further, but her heart-rate increased to 108 and the patient became weak. Her diet was therefore increased eventually to 1700 calories. Her general condition grew stronger, the pulse fell to 85, the pains returned somewhat, especially in knees, but her hands retained their objective and subjective improvement (loss of weight from 161 to 145 pounds). She was instructed to follow this compromise diet all summer. This case showed unquestionable and satisfactory response to diet, but her heart was unequal to carrying her through in the way desired.

Case XXXIII.—Dr. X., aged thirty-eight years, noticed in early January, 1915, a slight tenderness of the middle joints of some of the fingers of both hands. Toward the end of February there was added to this an ache involving also the lumbar spine. An *x*-ray, taken and interpreted by Dr. Henry Pancoast, director of the *x*-ray department of the University of Pennsylvania Hospital, showed inflammatory fog around the affected joints together with rarefication and overgrowths along the shafts of the bones, an interpretation shared by Dr. Newcomet. Being familiar with some of the writer's cases the patient attempted a diet, and in ten days lost six pounds. He had always been a large eater, particularly of starches and sugars. When seen by the writer he was given a mixed diet of about 1200 calories, and by eight or ten days subjective improvement was established. During an attack of influenza, which then began, he lost more weight, but after about six weeks of adherence to his diet he was entirely free from symptoms, and during August took part in a military encampment, by this time eating much more generously. The diet was carefully carried out in this instance, and the patient himself weighed the ingredients on scales. The loss of weight was about ten pounds in all, from 173 to 163.

Feeding Experiments. The following cases will be detailed at greater length, because they were the subjects of experiments in feeding which developed several points of interest. These experiments were conducted with the full understanding of the patients, who appreciated the possible importance of their coöperative efforts.

Case XXXIV.—Mrs. S., aged thirty-six years, referred by the courtesy of Dr. Frank Dickson, was admitted November 9, 1914, and discharged February 23, 1915. An arthritis of over seven years' duration, involved the jaws, shoulders, elbows, wrists, ankle, and knees; the hands slightly; the right knee was resected seven years

before. A probationary house diet yielded 1892 calories. On 1072 calories of mixed diet, she improved slowly but steadily, and on January 9, being up and out daily and feeling well, she was given the diet presently to be described.

It should be here stated that for some time the writer had been following the hydrogen ion concentration in the urine of joint cases at various stages of their progress, but no definite conclusions could be reached. The variations of diet in the same and different cases seemed apparently to mask any reaction or fluctuations due to the disease itself, and it was not until the appearance of Blatherwick's "Studies"[3] on the nature of the ash in various foods and its influence upon urinary acidity that these relations became clear and a standard diet could be applied. Accordingly in the following cases one of the diets there described has been utilized as a basis for the study of the urinary acidity of joint cases in health or disease, and also when the influence of certain foods was tested. These standard diets were made up equivalent in caloric value to the mixed diets under consideration, and were chosen without other regard to therapy. The patient was therefore given graham crackers, 75 gms., butter, 36 gms., milk, 720 c.c. per diem = 1009 calories. This was continued for ten days to allow her condition under it to be watched and the urinary acidity to be followed while she was free from symptoms. The basis upon which all the cases in these series have been treated is, in part, that the carbohydrates form at least one of the factors productive of joint disease, and favorable results have followed their curtailment. Other evidence pointed the same way, but the "experimentum crucis" had not been made of inducing the symptoms of the disease by means of pure carbohydrate in a patient previously rendered free from them—a reinduction of the disease, in short.

On the afternoon of January 19, therefore, and twice daily thereafter, the patient was given 62 gms. of "candy" made from cane-sugar, water, a little oil of peppermint to flavor it, and one teaspoonful of vinegar to 528 gms. of sugar. This gave 66 gms. of sugar at each feeding, or 264 calories twice a day, a total ingestion of 528 calories in addition to her regular diet. The total for twenty-four hours was therefore 1089 plus 528, or 1617 calories, an amount somewhat less than the house diet on admission, but considerably greater than that upon which she had improved. It is to be remembered that at this point she was practically free from symptoms, up all day and taking exercise on the porch. The abbreviated notes of her progress follow:

January 22. One joint of left hand shows a slight sensitiveness not present for at least a month.

January 23. Pain increased and present in shoulders and wrist.

[3] The Specific Role of Foods in Relation to the Composition of the Urine, N. R. Blatherwick, Archives Int. Med., September, 1914, xiv, 409–450.

January 24. Hand slightly better. Left shoulder worse. Painful red swelling with effusion just above left elbow. Not sore for six weeks.

January 25. Thumb better but still sore. Elbow and shoulders as before. Jaw on the left painful. Patient is very uncomfortable.

January 26. No candy given today. Jaws worse, both sides equally. Shoulders better. Thumb and knuckles about the same.

January 26. Better as a whole. Jaws, shoulders, and thumb ache less. One joint of left hand has ceased to hurt. Pressure over seventh cervical vertebra elicits soreness and suprascapular muscles on left ache. Undoubted improvement.

January 28. Muscles of back and seventh cervical vertebra are the chief sites of pain. Today patient returns to the mixed diet of 1089 calories, taken before the graham-cracker diet, as the latter has become very monotonous.

January 29. About the same.

January 30 and 31. Better.

February 2. Improvement continues. Muscles of back and neck and the left elbow show some residual soreness. Progress from this date was entirely satisfactory and continued so. Last seen December, 1915, when improvement was maintained. Loss of weight in hospital from 158 to 143 pounds. Comment on this experiment will be made later.

Case XXXV.—James N., aged forty-four years, referred by the courtesy of Dr. T. T. Thomas, was admitted November 12, 1914, and discharged March 23, 1915. An arthritis of four years' duration, involving shoulders, elbows, wrists, hands, hips, knees, and ankles, incapacitated him. He made noticeable progress on a house diet of 2170 calories, and after a variety of diets of lower caloric value from 1512 to 1829, made steady progress, subject to occasional exacerbations of an acute nature, generally with some effusion. During three months these became less severe and he recovered from them more rapidly, being increasingly free from chronic symptoms in the interim. It was obviously necessary to wait until these exacerbations had become negligible and until observation of the case over a long period of convalescence had shown its true course before a feeding experiment comparable to the last could be attempted. After the patient had been given six or seven different diets, had been watched through six decreasing exacerbations of his chronic arthritis, and was free from pain anywhere in his body, it was thought, on February 16 1915, that a repetition of the carbohydrate feeding would be justified and that any clear response could be ascribed to it. He was accordingly put on 1827 calories, consisting of graham crackers, 100 gms., butter 10 gms., one cup of weak tea with 7 gms. sugar, and one apple of 150 gms. three times a day. For six days he had had practically the same thing, except that 360 of the calories

had been supplied by three glasses of milk per diem; but it was thought advisable to remove, as far as possible, all protein from the diet so that any disturbance referable to the food intake must be due to some other factor in it. Prior to this he had been taking 1512 calories of a mixed diet. A difficulty to be here considered, which had been operative in certain earlier indefinite experiments, was that the patient's total caloric tolerance seemed to be pretty high, as evidenced by the house diet of 2170 calories, on which he showed improvement. This must be exceeded considerably by the sugar calories to have any effect, and such an addition might be physically and psychically difficult of accomplishment. There is a very definite limit to the amount of pure carbohydrate one can willingly take for any length of time. However, beginning with February 16, he was given twice daily 138 and then 150 gms. of candy, made as described in the first case, but without any vinegar. This was equivalent to 150 gms. of cane-sugar, or 600 calories. The patient professed at first to like this and ate it readily, but later did so with great difficulty. The notes of his progress follow.

February 17. Says he feels a little bit stiff in both shoulders, knees, and feet.

February 18. Feels somewhat "tired." Shoulders and knees slightly stiff, but very little so.

February 19. About 7 P.M. yesterday began to feel pain and stiffness in left shoulders, after which pain developed in feet and other joints. Today he has pain on active or passive motion or on pressure at left shoulder, both wrists, between the first and second metacarpophalangeal articulations of left hand, at right thumb (slightly), at second metacarpophalangeal joint of left hand (which hand cannot be closed as usual), at midjoint of middle finger of left hand, and near internal malleoli of both ankles. He cannot rise on his toes as well as usual and does so with considerable pain. Is quite wretched and could not be kept on this diet any longer. There is possibly some fluid in midphalangeal joint of midfinger of left hand. Same point on right hand is slightly sensitive to pressure. "Jumps" visibly if a sore point is touched. Ate no supper last night or breakfast or dinner today. Candy to be stopped. Has had none since 4 P.M. yesterday.

February 20 Much better. Points still showing tenderness are wrists and hands, which are much improved. Took one meal of graham-cracker diet yesterday and all three today.

February 21. A trace of tenderness in one joint only.

February 22. Feels very well.

February 23. Diet changed to a mixed one of 1512 calories.

This patient's further progress was uneventful, and he went home feeling entirely well on March 23, except that on March 3, under some rather forced mixed feeding to make up for the loss of

weight sustained during this last attack, patient developed some toothache, a red throat, some joint discomfort, and fever, the latter up to 100⅗°. He had been losing weight or remaining stationary, but as the result of this feeding had gained five pounds in all. His diet was again sharply cut and he improved at once. The patient was still well six weeks after leaving the hospital, and though he deliberately departed from his diet after July, and gained 20 pounds, he was in very fair shape in November, 1915.

The results in this case seemed pretty clear. There could be no question of his original illness; of his marked convalescence; of the exacerbation under the sugar feeding; of his prompt convalescence when the sugar was removed (hastened, possibly, by his refusal of all food for nearly twenty-four hours), or of the excellence of his condition when he left the hospital. The only doubt that can enter is, whether there could have occurred a severe exacerbation coincident with the experiment, such as those noted early in the course of treatment and referred to above. The diminishing nature and frequency of these attacks, their practical cessation under strict diet and their somewhat different character make this unlikely.

Case XXXVI.—Miss C., aged thirty-five years, referred by the courtesy of Dr. E. H. Goodman, came under observation February 1, 1915, and was discharged July 7, 1915. An arthritis of four years' intermittent duration involved the jaws, shoulders, elbows, hands, knees, and ankles. The patient had wide-spread and severe psoriasis, involving nearly every part of the body. Treatment of this case developed several points of interest. Her previous treatment had included vaccines and radium water. She began to improve on a house diet of 1718 calories. On February 18 she was given a diet of 75 gms. of graham crackers, 1 apple, 5 gms. butter, and 9 gms. sugar at each meal, yielding 1410 calories a day, with the idea of maintaining the joint improvement and at the same time helping the psoriasis along lines indicated by the interesting work of Schamberg, Ringer et al.[4]

These authors have shown that there is in psoriasis a strong tendency toward the retention of nitrogen and that a curtailment of the nitrogen intake to a minimum may be followed by great improvement or cure. By March 16, 1915, it was evident that the psoriasis had improved about 75 per cent., but that the joints, after having made substantial progress and changed their shape, were "hanging fire." She was then put upon a mixed diet of 1002 calories, on the possibility that the carbohydrate intake was too high. After eight days she had lost 9 pounds *in toto* from the start, her weight being 110 pounds. It was thought that a still more restricted diet was indicated, but that preparatory to putting

[4] Research Studies on Psoriasis, Schamberg, Kolmer, Ringer, and Raiziss, Jour. Cutan. Dis., October and November, 1913.

her upon it an effort should be made to increase her weight and strength. This was a mistake, but she was put back, March 24, 1915, on a fuller diet, very high in carbohydrates but low in nitrogen, and consisting of bread, potatoes, rice, etc. The hope was that the psoriasis could be held in abeyance while her weight was increased even at the expense of her joints, but she relapsed so promptly and severely that on March 30, 1915, the psoriasis was everywhere violent and the arthritis as bad as it had ever been, or worse. It was felt that radical measures alone would do her any good, and she was put upon a graham-cracker diet as above of 846 calories. The joints responded at once, objectively as well as subjectively, and by April 11, 483 calories of fat had been added in the shape of olive oil and butter, with the idea of increasing her weight, which had gone down to 103 pounds, and also of testing out her tolerance for fat as compared with carbohydrate. Her weight reached 101 and then began to climb. The psoriasis showed no real improvement this time on the low nitrogen diet, though the nitrogen intake was lower than before, and at her request she was given, by Dr. F. C. Knowles, on April 13, an ointment containing liquor carbonis detergens and salicylic acid, after which the psoriasis improved. Her further progress was steady, and by April 21 she was taking 2054 calories, of which only 860 came from the graham crackers and apples. On June 14, 1915, the woman was practically well of both her psoriasis and joint disease, save for a very trifling sensitiveness on lateral pressure over the middle knuckle of the right midfinger. The ointment had been stopped on June 2. She had often previously been on full doses of the salicylates without benefit, and was two weeks convalescent as to her joints when the ointment was started.

It was thought that this would be a good case in which to try the effect of high carbohydrate feeding, as in the last 2 cases, so on June 20, all fat was substituted by pure carbohydrate in the form of peppermint candy. She was getting a total of 2053 calories, of which 1207 came from fat and whisky. This was substituted by 300 gms. sugar, equivalent to 1200 calories, but as no injury was apparent on the 24, she got a further addition of 40 gms. cornstarch and 35 gms. sugar, equivalent to 300 calories, making the total 2353. On June 26 she was still well, however, except for a bad headache, and could not force down her food, so the experiment was stopped. She was put back on the high fat diet, substituting saltines for graham crackers, and left for home July 6, apparently perfectly well. Before the forced carbohydrate feeding her weight had come up under the fat to 105, and when she left it was 109 pounds. It is hard to escape the view that the psoriasis was a complicating factor in this case. The caloric reduction was almost the lowest the writer had ever reached in treatment, and shows the freedom from danger, under properly

guarded conditions, of such extremities if they become necessary. No exacerbation of her disease was caused by the high carbohydrate feeding within the period covered. What might have happened had the diet been continued cannot be said, though it is noteworthy that the next case to be described behaved analogously for the same period of time, and then developed a sharp exacerbation while still under the forced feeding. It is possible that the strong tendency toward retention of nitrogen which psoriatics show was a factor which added to the difficulty of treatment. It should be added that joint cases which recover along these lines acquire in most instances an increased tolerance for food if they adhere carefully to their regime. This is beyond question, and is possibly an added reason for the tolerance shown in the experiment last recorded, since she had been upon a limited intake of carbohydrate for twelve weeks, and was almost perfectly well at the time. Properly to test the effect of carbohydrate her maximum intake when well should have been exceeded by a much greater margin, but this was hardly possible of accomplishment. On January 21, 1916, this patient was apparently in robust health, weighing 122 pounds, eating much more generously, and walking on some days as much as eight miles. It is interesting to note that throughout her illness at the hospital she had, and still has, a carious molar tooth, which cannot be ignored as a possible source of infection. Her recovery in spite of this is in keeping with other such instances already cited.

Case XXXVII.—Fred M., aged thirty-nine years, referred by the courtesy of Dr. A. H. Gerhard, was admitted April 24, 1915, and discharged July 9, 1915. An arthritis of fourteen months' duration involved the shoulders, elbows, wrists, hands, knees, and feet, and incapacitated him. A probationary house diet yielded 2078 calories. He was given a mixed diet of 1268 calories, on which he showed slight improvement. This was changed on May 15, 1915, to 1065. He developed tonsillitis on May 25, after showing evident improvement, and was held back somewhat. By June 15 he was in very good shape, and a feeding experiment with carbohydrate was begun. He was put on a diet of graham crackers 60 gms., apple 150 gms., and weak tea with 7 gms. sugar, three times a day, yielding 1072 calories. On June 20 he got 322 gms. of candy, equivalent to 300 gms. of cane-sugar, distributed during the day. This added 1200 calories, making a total of 2272. On June 24, 40 gms. of cornstarch and 35 gms. of cane-sugar were added to the intake daily, cooked with water and a little vanilla, and making a bulk of 546 gms. This yielded in all 2572 calories. The notes follow:

June 26. For the first time since the forced carbohydrate feeding he seems to have more pain, chiefly in both wrists. Left knee slightly stiffer.

June 27. Says he felt worse when he woke up today than for some time past. At 5 P.M. has increased pain in both wrists, left little finger, left thumb, left ankle, shaft of left arm, and left knee.

June 28. Worse. Points of soreness are right side of chest posteriorly, right side of neck, right knee, left shoulder, both wrists (worse than yesterday), left forefinger, left little finger, left knee, and both ankles.

June 29. Worse. Additional points are the right shoulder, toes, and left pectoral muscles. Diet cannot be pushed further. To return to simple graham-cracker diet tomorrow.

July 1. Has made undoubted progress and is better considerably today at eleven points.

July 3. Right and left little fingers and both wrists are somewhat worse. Right knee shows effusion. Other points are better or well.

July 5. Effusion less. Distinctly better elsewhere.

July 8. Progress satisfactory but not yet back to his condition before the experiment. He grew very tired of the diet and was returned, on July 7, to a mixed diet of 1072 calories. Sent home with instructions to follow the diet for the rest of the summer and report in the fall.

In this case there could be no doubt of the exacerbation induced by the forced feeding. The man was made almost worse than he had been at any time. Also, he had had no prior exacerbations of a nature to be confusing. It is interesting to note, as mentioned in the last case, that these two cases happened to be carried through on exactly the same dates, and that the present case had hardly begun to have symptoms—had certainly shown none that were definite—by the date upon which the former had to be taken off her forced diet without having shown an exacerbation.

Reference is omitted to several other cases under treatment at the present writing.

Observations on the Hydrogen Ion Concentration of the Urine and the Carbon Dioxide Tension in the Alveolar Air. As already referred to in the text, observations have been conducted in some cases of this series on the hydrogen ion concentration of the urine, as a measure of the true urinary acidity. The methods followed were those developed by Henderson and Palmer,[5] and depend upon the comparison of diluted urine, to which an indicator has been added, with a series of flasks of prepared solutions. These solutions contain weighed amounts of the sodium and potassium salts of phosphoric and acetic acids, and correspond to definite concentrations of the hydrogen and hydroxyl ions, which, in terms of physical chemistry, express acidity and alkalinity. The notation used signifies the logarithm of that number which

[5] Clinical Studies on Acid Base Equilibrium and the Nature of Acidosis, Walter W. Palmer, Lawrence J. Henderson, Archives Int. Med., August, 1913, xii, 153–170.

expresses the actual acidity or alkalinity. The logarithm decreases as the acidity increases, and *vice versa*, and the minus sign is here omitted.[6]

A study of the figures shows that until the patients were placed upon the standard graham-cracker diet no characteristic reactions could be discerned, although the urines in disease and convalescense were contrasted. Cases in apparently comparable stages of the disease gave varying results, and the same case at the same period sometimes showed variations from marked acidity to marked alkalinity.

The samples were from twenty-four-hour specimens, preserved with toluol and kept on the ice. Doubtful ones were discarded. Occasionally, "stat" specimens were also examined. Space will not permit of giving the figures for the cases which were not placed upon the standard graham-cracker diet.

The first case examined upon the standard diet was in Case XXXV. The urine showed the same indefinite variation on the probationary house diet, and when the patient was convalescent on it, but when he was convalescent on a constant mixed diet it approximated stability of reaction. Around January 10, it showed an interesting drop from an acidity of 6 or thereabouts to one of 7.7, a difference of 4.5 in the scale of eleven flasks, from which it slowly recovered. Coincident with this was an exacerbation of his arthritic condition, the urinary change slightly anticipating the attack. This was repeated sufficiently often in this and other cases to suggest that it is a feature of such disturbances, and reflects a metabolic change whose nature is not yet clear. In two instances it led to the anticipation of otherwise unsuspected oncoming attacks, but sometimes appeared immediately afterward. These changes can be seen by consulting the appended tables, and occurred as follows: Case XXXV, as recorded above and again accompanying an exacerbation on January 24. Again on February 19, accompanying an attack produced by carbohydrate feeding. Case XXXIV, on January 11, accompanying an ephemeral exacerbation in one hip and about January 26, accompanying an exacerbation induced by carbohydrate feeding. There was one isolated drop in the acidity on January 6, which was accompanied by no noteworthy symptoms and cannot be explained. It may have been due to an error in the collection.

Case XXXVI showed a less constant reaction during the period observed on the basal cracker diet, owing probably to the error of feeding asparagus which necessarily varied in quality, water

[6] It should be here stated that the logarithmic figures do not portray graphically the differences in reaction of the urine, as a drop from No. 7 to No. 4 in the scale of eleven flasks, three members of the series, is indicated merely by the change from 6 to 7 in the logarithm. This should be borne in mind by those unfamiliar with the notation.

content, etc., and it is interesting to note that the high carbohydrate feeding *per se* made no great change in the hydrogen ion concentration during the period observed. This is in keeping with the observations of Blatherwick on the effect of the ash in diets, and additionally suggests that the change in the cases of induced exacerbation depended upon the systemic disturbance. Owing to the failure to induce an exacerbation the figures are omitted to save space.

Case XXXVII showed the same reduction beginning around January 25, coincident with an attack induced by forced carbohydrate feeding. In the cases above mentioned, the reaction of the urine did not always return at once to its original level but remained alkaline or less acid for some time. The reaction of the urine depends chiefly upon the relative amounts of the dimetal hydrogen phosphate and the monometal dihydrogen phosphate present. A decrease in the acidity as here found represents in the urine an increase of the dimetal (disodium or potassium) hydrogen phosphate as compared with the monometal (monosodium or potassium) dihydrogen phosphate. The metabolic cause of this urinary change remains a subject for future investigation.[7] The long period required to bring these patients into the convalescent or recovered condition, together with the further period and coöperative difficulties incidental to the induction of a clear exacerbation, seem to warrant the presentation of these cases now.

The urinary findings need corroboration from a longer series before they can be accepted as established and observations are pending to that end.

Case XXXV.—James N. November 17, 1914. Log. = 6.7. Convalescing on house diet averaging 2170 calories.

November 17. Log. = 5.7. "Stat" specimen. Noon: house diet.

November 18. Log. = 6.3. House diet.

November 18. Log. = 7.4. "Stat" specimen. Noon: house diet.

January 5, 1915. Log. = 5.7. 1663 calories of mixed diet; three days convalescent from a "flare up" in the course of recovery.

January 6. Log. = 5.3. 1663 calories of mixed diet. Very well.

January 7. Log. = 5.7. 1683 calories of graham crackers and milk. Well.

January 8. Log. = 6. 3650 c.c. ditto.

January 9. Log. = 6. 2485 c.c.

January 10. Log. = 6.3. 2100 c.c.

January 11. Log. = 7. 1530 c.c.

January 12. Log. = 6.7. 1120 c.c. Exacerbation on night of January 11, on Blatherwick's diet.

[7] Since the completion of the above work, Wilson, Stearns and Janney have reported an alkalosis after parathyroidectomy in dogs. Journal of Biological Chemistry, November, 1915.

January 13. Log. = 7.7. 910 c.c. Still sick and effusion in knee.

NOTE.—To test the effect of varying the volume as a possible contributory cause of this difference, the urines of Cases XXXV and XXXIV were diluted in varying amounts up to 100 and 300 per cent. greater volume respectively, but gave the same figures. Except where specified, the specimens were taken from twenty-four-hour collections.

January 14. Log. = 6.3. 1980 c.c.

January 15. Log. = 5.85. 1800 c.c. Well.

January 16. Log. = 5.85. 1840 c.c. 2649 calories started yesterday.

January 17. Log. = 6.3. 2190 c.c. 1827 calories started yesterday at lunch. Now on low proteid diet of graham crackers.

January 18. Log. = 6.2. 1080 c.c.

January 19. Log. = 5.85. 1960 c.c.

January 20. Log. = 6. 2390 c.c.

January 21. Log. = 6. 1870 c.c.

January 22. Log. = 5.85. 1110 c.c.

January 23. Log. = 6.85. 1780 c.c. Well.

January 24. Log. = 6.5. 2160 c.c. Exacerbation last night.

January 25. Log. = 7. 1790 c.c. Effusion in knee still left.

January 26. Log. = 7. 2000 c.c. Well; some effusion still left.

January 27. Log. = 7. 1930 c.c. Well.

January 28. Log. = 7. 2300 c.c.

February 14. Log. = 6.3. 1810 c.c. Graham-cracker diet 1827 calories. Very slight attack in one joint. Began 6 A.M. Gone by noon, almost.

February 15. Log. = 6. 1530 c.c.

February 16. Log. = 6.15. 1810 c.c.

February 17. Log. = 6.7. First urine after candy feeding. 1820 c.c.

February 18. Log. = 6.3. Below par.

February 19. Log. = 7. 1100 c.c. Sharp attack. No effusion.

February 20. Log. = 6.85. 700 c.c. No breakfast or dinner yesterday. Candy stopped yesterday.

February 21. Log. = 5.85. 660 c.c. Practically well.

February 22. Log. = 5.85. 1600 c.c. Well.

February 23. Log. = 6. 1620 c.c.

March 6. Log. = 5.7. 2420 c.c. Mixed diet of 1512 calories started February 23.

March 7. Log. = 5.7. 1880 c.c.

March 8. Log. = 5.6. 1360 c.c. Fever today; joints worse; patient caught "cold."

March 9. Log. = 5.85. 1730 c.c.

March 10. Log. = 5.5. 1590 c.c.

CASE XXXIV.—(Mrs. S.)—November 17, 1914. Log. = 5.7. House diet averaging 1892 calories.

November 17. Log. = 5.7. "Stat" specimen at noon.
November 18. Log. = 6.
November 18. Log. = 5.2. "Stat" specimen.
January 5, 1915. Log. = 5.7. Well.
January 6. Log. = 7.
January 8. Log. = 5.85. 410 c.c.
January 9. Log. 6.5. 800 c.c. Comparison not good. Exacerbation in hip.
January 10. Log. = 5.85. 700 c.c. Diet of 1089 calories of graham crackers and three glasses of milk begun today at breakfast.
January 11. Log. 7.7. 1000 c.c. Exacerbation in hip better.
January 12. Log. = 5.3. 380 c.c. Well.
January 13. Log. = 5.85. 330 c.c.
January 14. Log. = 5.5. 460 c.c.
January 15. Log. = 5.5. 300 c.c. Well.
January 16. Log. = 5.5. 420 c.c.
January 17. Log. 5.5. 410 c.c.
January 18. Log. = 5.5. 520 c.c.
January 19. Log. = 5.5. 410 c.c. Candy started with 66 gms. of cane-sugar b. d. 528 calories per diem; total calories 1617.
January 20. Log. = 5.7. 610 c.c. Menstruating.
January 21. Log. = 5.85. 630 c.c.
January 22. Log. = 5.5. 280 c.c. Costive.
January 23. Log. = 5.7. 530 c.c.
January 24. Log. = 5.7. 315 c.c.
January 25. Log. = 6. 530 c.c.
January 26. Log. = 5.85. 350 c.c. Induced exacerbation.
January 27. Log missing. 510 c.c. Candy stopped on January 26.
January 28. Log. = 6.7. 660 c.c. Better, but distinctly sore. Effusion in elbow bursa.
January 29. Log. = 5.85. 550 c.c.

Case XXXVII (Fred M.).—June 16, 1915. Log. = 5.5. 500 c.c. On graham-cracker diet of 1072 calories. Urine scanty and full of urates.
June 17. Log. = 5.5. 810 c.c.
June 18. Log. = 5.7. 460 c.c.
June 19. Log. = 5.6.
June 20. Log. = 5.5. 300 c.c.
June 21. Log. = 5.5. 310 c.c.
Beginning yesterday got 300 gms. of cane-sugar daily, 1200 calories, or 2272 calories in all.
June 22. Log. = 5.6. 360 c.c. Not quite so well.
June 23. Log. = 5.8.
June 24. Got additional 300 calories from cornstarch and cane-sugar.
June 25. Log. = 5.8. 350 c.c.

June 27. Log. = 5.85. 280 c.c.
June 28. Log. = 6.3. 450 c.c. Exacerbation.
June 29. Log. = 6.2. 570 c.c.
July 1. Log. = 6.4. 340 c.c. Somewhat better.
July 2. Log. = 6.4. 340 c.c.
July 3. Log. = 6.4. 320 c.c. Worse.
July 4. Log. = 6.2. 320 c.c. Slightly better.
July 5. Log. = 6.3. 320 c.c.
July 6. Log. = 6.2. 315 c.c. Doing well.

In view of the urinary changes recorded above, and with a view to throwing light on a possible acidosis as well as for other reasons, observations were conducted on the carbon dioxide tension of the alveolar air in several of these cases during ill health and convalescence.

Higgins's[8] adaptation of the Plesch method was employed, using 1000 c.c., as suggested by Boothby and Peabody.[9]

The analyses were made with a Tutweiler mercury gas burette as modified and used by the United Gas Improvement Company of Philadelphia.[10]

Comparisons were first made of the results obtained by using 1000 c.c. of air, and by the exhalation method, the former being finally selected as less variable, though the difference was not great, as pointed out by the above authors.

As seen by the accompanying tables the normal cases undoubtedly averaged slightly higher than the arthritis cases, but while this is suggestive, the difference is not greater than may possibly be accounted for by the fuller diets of the normal cases. Further observations are pending on this point. It is interesting to note, however, that T. K., with active arthritis and on a house diet, had low figures. Fred. M., Case XXXVII, while convalescing on 1072 calories of a graham-cracker diet, averaged less than 32.06 mm., and during an induced exacerbation getting 2572 calories daily of nearly pure carbohydrate, gave an average of only 40.5 mm.

Case XXXVI, Miss C., gave low figures throughout, although at the time getting 2054 calories, supplied chiefly by graham crackers, fat, and whisky. When getting nearly 1000 calories more than Case XXXVII she gave even lower figures than he did. It is evident, therefore, that the conclusion to be drawn at the present writing is that there is no great change of the alveolar carbon dioxide tension characteristic of rheumatoid arthritis. There may be one of minor degree, but further observations are necessary to establish this point.

[8] The Influence of Food, Posture and Other Factors on the Carbon Dioxide Tension in Man, Am. Jour. Physiol., April 1, 1914, xxxiv, No. 1.

[9] A Comparison of Methods of Obtaining Alveolar Air, Archives Int. Med., March, 1914, xiii, 497–506.

[10] The writer wishes to express his marked appreciation to Mr. George H. Fulweiler, chief chemist of the United Gas Improvement Company, for courtesy and assistance in this connection.

OBSERVATIONS ON THE CARBON DIOXIDE TENSION OF THE ALVEOLAR AIR.

Normal Cases.

R. P.:			
May 14, 1915.	41.60 mm. Hg.	12.30 P.M.	Four hours after breakfast.
May 17, 1915.	41.20 "		
May 18, 1915.	39.41 "	3.15 P.M.	
May 18, 1915.	41.20 "	4.30 P.M.	
June 2, 1915.	39.63 "	7.00 P.M.	Lunch at 2 P.M.
June 3, 1915.	42.17 "	10.30 A.M.	Breakfast at 8.30 A.M.
Michael:			
June 9, 1915.	43.08 "	5.00 P.M.	Dinner at 11.30 A.M.
	45.35 "		
J. F.:			
June 15, 1915.	39.66 "	6.10 P.M.	Lunch at noon.
Dr. L.:			
June 16, 1915.	39.1 "	6.00 P.M.	No food since 1 P.M.
June 16, 1915.	41.00 "	6.25 P.M.	No food since 1 P.M.

Arthritic Cases.

T. K.—In active arthritis and on house diet:

June 2, 1915.	34.21 mm. Hg.	5.30 P.M.	Dinner at noon.
June 3, 1915.	32.94 "	11.00 A.M.	No breakfast.

Took 200 gms. cane-sugar and juice of one and a half lemon in water at noon.

	35.03 mm. Hg.	12.15 P.M.	
	36.92 "	12.40 P.M.	
	35.13 "	1.05 P.M.	
	36.37 "	1.30 P.M.	
	36.01 "	1.50 P.M.	
	36.88 "	2.35 P.M.	
	38.02 "	3.20 P.M.	
	37.36 "	3.50 P.M.	
	38.45 "	4.20 P.M.	
June 4, 1915.	37.63 "	4.50 P.M.	Dinner at 12.30.
June 5, 1915.	36.62 "	10.55 A.M.	No breakfast.

Case XXXVI.—Miss C. Doing well. Getting 2054 calories of graham crackers and fat.

June 11, 1915.	30.44 mm. Hg.	4.00 P.M.	No food since 12.15 P.M.
	32.86 "	5.00 P.M.	
	29.05 "	5.40 P.M.	
June 15, 1915.	30.39 "	4.30 P.M.	Lunch at 12.30 P.M.
	28.72 "	5.00 P.M.	
June 19, 1915.	33.10 "	9.40 A.M.	No breakfast.

Average of two experiments.

Case XXXVII.—F. M. Pretty well on 1072 calories of graham crackers, etc. Some traces of trouble left yet.

June 17, 1915.	29.39 mm. Hg.	4.50 P.M.	Lunch at 12.30 P.M.
	34.74 "	5.15 P.M.	Breathing too slowly and result too high.
	32.05 "	5.40 P.M.	
June 19, 1915.	35.35 "	11.00 A.M.	Before breakfast.
June 28, 1915.	41.17 "	4.00 P.M.	No food since lunch, but is on the forced carbohydrate diet of 2572 calories.

Distinctly worse:

35.95 "	4.40 P.M.	
40.38 "	5.05 P.M.	

In the studies previously reported were included observations on the urea and non-coagulable nitrogen of the blood in rheumatoid arthritis which gave entirely normal values. In view of the opportunity, however, to make comparisons in this respect between health and disease, observations were begun to that end, but only one was completed in time to be published. Blood was therefore analyzed from other cases, first before treatment and then when they were convalescent or had recovered under treatment.

The first 3 were ill and no opportunity was afforded to get the blood a second time as they left the hospital, but the figures are given as showing further the normal values. The conditions were as previously reported. It is seen that there is no appreciable difference in the blood findings during active arthritis and those taken during the convalescent or recovered period. The analyses were made by Dr. Charles W. Lüders, to whom acknowledgment is due. Since the completion of this work, Folin and Dennis have reported[11] analyses of uric acid and non-protein nitrogen in 5 cases of arthritis deformans among a larger series of gouty and other patients. These writers conclude that: (1) "In gout the blood is almost invariably abnormally high in uric acid, while the other waste products represented in the non-protein nitrogen of the blood are usually within the normal limits. In arthritis also the blood is not infrequently abnormally high in uric acid but most such cases have abnormally high non-protein nitrogen as well. (2) Neither qualitative tests for uric acid in the blood nor quantitative determinations of the uric acid alone can be depended on in the differential diagnosis of gout and other joint diseases. (3) For a differential diagnosis in doubtful cases of gout or arthritis by means of blood analyses the patient must be on a purin-free diet and uric acid determinations must be accompanied by determinations of the non-protein nitrogen (or urea)."

In their series only 1 of the 5 cases of arthritis deformans gave very high values for non-protein nitrogen (104 mgs.) the others being in the neighborhood of 60 mgs. per 100 c.c. blood, but as the figures of these careful workers are at variance with those of the present writer and imply a means of differentiating doubtful cases of rheumatoid arthritis from doubtful cases of gout, it seems well to include here, with the new cases, those already reported, since they illustrate the great uniformity and low values of the findings in a total of 9 cases of rheumatoid arthritis. These 9 instances were in relatively young individuals free from complicating diseases. They all represented clear-cut examples of rheumatoid arthritis and on 3 of them the analyses of ill health were repeated during recovery with practically identical results. These observations were conducted by two sets of observers, and there seems hardly

[11] Archives Int. Med., 1915, No. 1, pp. 16–33.

NEW WORK JUST READY

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room for the possibility of error in the technic. The simplest explanation of this discrepancy is that the cases studied by Folin and Dennis may have had some complicating factor.

Old series.	Total non-coagulable nitrogen in mg. per 100 c.c. blood.		Average.	Urea in mg. per 100 c.c. blood.		Average.
Case VIII. Patient ill . .	20.2	21.7	21.00	11.2	12.6	11.9
Case IX. Experiment 1. Patient ill	27.8	25.7	26.00	14.7	14.5	14.6
Case XI. Patient ill . .	22.6	22.7	22.65	9.3	10.2	9.6
Case IX. Experiment 2. Patient convalescent . .	27.0	26.4	26.70	12.8	..	12.8
Case XIII. Patient ill .	24.6	25.5	25.00	12.0	14.2	13.1
Case XII. Patient ill . .	24.4	25.0	24.70	10.4	..	10.4

New series.	Total non-coagulable nitrogen in mg. per 100 c.c. blood. Average.		Total urea in mg. per 100 c.c. blood. Average.
Mrs. B. Ill	21.80	..	16.10
Mrs. S. (Case XXVI). Well	32.30		
Mrs. C. (Case XXVII). Improving	26.30		
Mrs. S. (Case XXXIV). Ill	28.70	Ill	12.37
Mrs. S. (Case XXXIV). Well	26.20	Well	14.70
James N. (Case XXXV). Ill	26.07	Ill	12.35
James N. (Case XXXV). Well	28.30	Well	15.70

DISCUSSION. A review of the foregoing permits of certain general conclusions. In the first place it is plain from the end-results of Series 1 that cases of rheumatoid arthritis treated along the lines of dietary curtailment, as indicated, will, by and large, indefinitely retain the improvement which may have occurred, provided they adhere to the general principles already followed. A number of cases have now been followed for a long time after recovery, the longest period being nearly five years.

In the second place, it is plain from an extension of the method to another and longer series that a large proportion of cases will show a response to treatment, there being 1 instance (Case XXX) in the 37 here recorded where no influence could be observed. In a definite proportion of cases so responding, treatment may be limited by intercurrent conditions and the desired end-result may be modified or entirely prevented. It was prevented in 1 of the cases which responded and remained under observation (Case XXI).

There are also limitations in cases emaciated or bed-ridden, where a curtailment of diet is in itself contra-indicated. Such cases must be improved in nutrition before they can be successfully carried through. In other words, there is nothing in these dietetic measures *per se* which is harmful if carefully watched, but there are advanced cases of rheumatoid arthritis which have gotten to the point where

any serious disturbance of their routine is dangerous to their already precarious health.

In these cases the arthritis can generally be influenced, but perhaps only at the expense of their already broken health. The writer desires to emphasize this. Even in such extreme types, however, something may be accomplished, and the effort is often well worth while if these truths be appreciated. It is also possible that subsequent study may show more types of cases which do not respond at all to these measures, as already illustrated by Case XXX, and the present report aims to present nothing more than the statistics to date. On the other hand, there can now be no doubt of the entire success of these measures in a great number, perhaps the great majority of sufferers from this disease if they be taken in hand early.

The writer wishes to emphasize again that there is nothing here which controverts the general view that many cases are precipitated or perpetuated by foci of infection. If these foci can be removed, such a step is the easiest and quickest way out for the patient. Unfortunately this is not always possible and frequently failure attends repeated effort to that end. However that may be, the most interesting point in this connection is that *cases showing unmistakable and obvious sources of possible infection in teeth, tonsils, or elsewhere, may improve or recover completely upon a dietary régime.* This is now unquestionable. The real explanation is not at hand.

Too much emphasis cannot be laid on the precautions to which attention has elsewhere been called.[12] Furthermore only minute attention to detail will achieve results and repeated changes in the dietaries may be required.

The writer has suggested previously upon several counts that the large bowel is not chiefly or directly responsible for the symptoms, as has been maintained by some writers, and the experience with Case VII bears this out.

As has been elsewhere noted, several cases of the last series showed some beginning improvement upon the regular house diet. The reason is obvious. If the food tolerance of patients happens to be high, as is often the case, the limited intake of the house diet may be helpful. For example, the evening meal at the Presbyterian Hospital in Philadelphia yields on the average only some 400 calories, and consists of a slice or two of bread, a cup of tea or milk, and some stewed fruit. This house diet, however, is rarely sufficient to maintain improvement, as it is too uncertain; and after obtaining an idea therefrom, in these instances, the total calories have been put safely below that level and made to come from con-

[12] The Nature and Successful Treatment of Rheumatoid Arthritis, Internat. Clin., iii, Series 24.

stant sources. In Cases XXXV and XXXVI the improvement which had begun on the house diet was not maintained and changes of diet were necessary. In the limited space at disposal no reference can be made to all the dietary changes in the various cases. The use of calories as a measure of the amount of food is of course for that purpose only and is not meant to imply necessarily any change in the caloric needs of the body.

The after-treatment of these cases is often important. After the arthritis has subsided the weight may be much lowered, and at about that period the writer has been in the habit sometimes of giving cod-liver oil. It is important to maintain the food level fairly constant for some time and to make additions very slowly, watching the result of each one. Orthopedic exercises are generally indicated, but cannot, of course, be undertaken until the patient has become well accustomed to the new level of equilibrium or has begun to gain weight. The question of the low caloric level at which some of these cases have improved, and indeed continued to live, is a large problem in itself, and brief reference only can be made to it. When the writer first published his results, these diets seemed somewhat iconoclastic, but the experiences of Dr. F. M. Allen in the low feeding of diabetics has since shown how eminently safely this can be carried to even a much further point. There is reason to suspect that some cases of rheumatoid arthritis have a lowered necessity for food as compared with most individuals, or at least with the necessity standards as now accepted. Reference has elsewhere been made to the suggestive overlapping of the symptoms of acute inflammatory rheumatism and those in some cases of chronic arthritis. Illustration of this is to be seen in Cases XXIII and XXIV, as well as in some of the exacerbations of Cases XXXIV and XXXV. It is, of course, not to be understood that acute inflammatory rheumatism and rheumatoid arthritis are regarded as identical, but some symptoms of the former may occur in the latter condition, strongly resembling the redness, swelling, pain, and fever of the acute variety. The undoubted improvement shown under dietary measures strongly suggests a common factor in some of these instances.

No drugs were given in the present series except where stimulation was needed, and, on one or two occasions, where treatment was interrupted. It should be stated that the writer has seen a considerable number of other cases to which no reference is made (many of them referred by his colleagues) because they have not been suitable for treatment or have improved upon the removal of the infectious foci, which step has nearly always been advocated where possible. One case, referred by the courtesy of Dr. Wm. G. Spiller, presented a long-standing ankylosis of both hips. It could not be determined that any active process was present or that any specific benefit followed upon treatment, although the

patient chose to keep to his diet, and writes that he feels in better general health.

Case XXXIV was of the type in which the large joints chiefly were involved, and it is interesting to note, as in some others, that the same favorable results were obtained. Multiple fibrous nodules, easily mistaken at times for bony formations, were present in Cases XXVIII and XXIX, and showed striking resolution under treatment.

In chronic cases long upon a fixed diet, attacks of slight nausea, diarrrhea and the like may occur, requiring mild catharsis and temporary suspension of the diet. Such attacks are apt to be accompanied by exacerbations. Also, it sometimes happens that long-standing cases who may do well for as much as six weeks, require further curtailment of diet even after marked improvement has occurred.

It has seemed important to emphasize with the second series a few of the points already mentioned in order to define the limitations and possibilities in these measures. Many minor points have been omitted, but it is hoped that the general principles have been made clear enough to enable others to obtain results. The sequelæ of long-standing rheumatoid arthritis are such that they, rather than the actual arthritis, often present the greatest obstacles. It is to be remembered that the arrest of the causative arthritis does not undo the harm done or transform a cripple, and that even after the causative disease is no longer active there are many factors which may be productive of pains and disability. There is no short cut in the treatment of these patients and the lesson to be learned is to begin early in the disease, before deformity has occurred, at which period most gratifying successes may follow. It is also true that some long-standing cases respond surprisingly well and in such the causative arthritis may be arrested.

The results of the feeding experiments, in which out of four attempts upon patients rendered convalescent or well, exacerbations were induced in three by the ingestion of pure carbohydrate, seem pretty definite. The mechanism of this is not yet clear. It may prove referable to a bacteriological or other cleavage in the small intestine, but whether the process begins there and then or later, the end-effects are of a metabolic nature. Observations upon muscular exercise, not mentioned here, support this and also indicate pretty strongly that within certain limits and under certain conditions, a greatly increased food intake may be made possible by marked bodily activity, when this is permissible.

The one unsuccessful experiment with forced carbohydrate feeding above mentioned can be at least partly accounted for, as suggested under Case XXXVI. The human factor and the limited degree to which biological experiments can be pushed under these circumstances introduce great difficulties, and it is conceivable

that had it been possible to ignore them, the same results would have followed here as in the other three instances.

The principle which this article endeavors to point out is that rheumatoid arthritis is often referable to the dietary factors above indicated. The operation of this principle can be appreciated in a large majority of cases where the measures in question are applied and subsidence of the inflammatory process follows to a greater or less degree. This is independent of the question of complete restoration to health which is conditioned by various factors in which chronicity of the disease and its sequelæ play an important rôle. As a matter of fact, however, a gratifying proportion of cases can be materially helped, as the above records show, and, in many, arrest of the disease process may take place.

The writer would like to express his obligation in a number of directions: To Dr. Alonzo E. Taylor, for helpful criticism and the use of his laboratories; to Dr. W. S. Newcomet, director of the Roentgen-ray department, for a long series of skilful coöperative studies; to Dr. Damon B. Pfeiffer, director of the pathological laboratory, for his helpful attitude at all times, and to the writer's colleagues on the staff of the hospital, for the use of beds in their wards, and other kindnesses.

Much obligation is also due to Miss Caroline Milne, directoress of nurses; Miss Helen Wallace, chief dietician, and the nursing staff at the Presbyterian Hospital, on the last of whom has fallen the laborious work of preparing the weighed diets.

Conclusions. 1. The general conclusions already published as to the application of a restricted dietary in rheumatoid arthritis find confirmation within the limits indicated in the present series of 19 cases.

2. In cases rendered free, or nearly so, of acute symptoms by dietary means, exacerbations have been caused as an "experimentum crucis," by the ingestion of pure carbohydrate. Upon the withdrawal of this the symptoms have again subsided. The conclusion seems unavoidable that carbohydrate is a factor in the etiology and treatment of many cases of this disease.

3. Contemporaneously with natural or induced exacerbations of the disease there seems to be a tendency for the urine to show a decreased acidity, as measured by the hydrogen ion concentration.

4. Observations on the carbon dioxide tension of the alveolar air in these cases show no great departure from the normal.

5. The non-protein nitrogen and urea of the circulating blood in uncomplicated cases of rheumatoid arthritis, examined both during ill health and convalescence, fall well within the normal range.

THE SYNDROME OF COAGULATION MASSIVE ET XANTHOCHROMIE OCCURRING IN A CASE OF TUBERCULOSIS OF THE CERVICAL SPINE.

By Ralph S. Bromer, M.D.,

HOUSE SURGEON, ORTHOPEDIC SERVICE OF THE CHILDREN'S HOSPITAL, BOSTON, MASS.

(From the Orthopedic Clinic of the Children's Hospital, Boston, Massachusetts.)

The following case occurred on the orthopedic service of Dr. Lovett at the Children's Hospital. Through his kindness it is possible for me to report this illustration of a symptom-complex which has been little described in American literature. Its study may serve to shed some light in the future upon similar cases, seemingly obscure, in which an equally characteristic spinal fluid may be discovered.

Case Report.—R. B., male, aged three years; birthplace, Boston; hospital record, 5786.

Family History. Father, mother, and three other children living and well. Paternal uncle died of tuberculosis of the lungs. No children dead. Mother has had no miscarriages.

Past History. Third child, full term, easy labor. Breast-fed for eighteen months. Eczema at three months; cholera infantum at eighteen months; bronchopneumonia during winter. Has always been a weak, inactive child.

Present Illness. Began September, 1914. Parents noticed that the child commenced to hold his head in left-sided torticollis. The latter getting increasingly worse, has been accompanied by some feverishness and considerable irritability. Appetite good; bowels regular.

Physical Examination. A poorly-developed and nourished child, sick and feverish; head, otherwise normal, held in marked torticollis. Eyes: pupils equal and react to light and distance. No nasal discharge. Throat clear. Teeth in good condition. Chest: fair expansion; no abnormal changes in breath sounds nor in percussion note. Heart: area is normal; sounds of good rhythm, clear, and loud. Abdomen normal, no masses.

Local History. Patient stands with a marked left cervicodorsal, right dorsal, and left dorsolumbar scoliosis, with the head tilted to the left and the chin rotated a little to the right. Walks with a very unsteady gait. Lying in bed no facial asymmetry is noted. Considerable elevation of the right shoulder and the inferior angle of the left sternocleidomastoid muscle, especially at the sternal attachment; no contraction of the trapezius. Head held in approximately 60 degress of left lateral flexion, 40 degrees of anterior

flexion, and 30 degrees of rotation to the right. All joint motions elsewhere throughout the body are free. General signs of old rickets, rather square head, enlarged epiphyses at elbows and knees, prominent abdomen, some outward bowing of the femora and tibiæ, slight Harrison's groove, and rachitic rosary.

Reflexes: Knee-jerks present on both sides. No clonus; no Babinski; no Oppenheim.

Von Pirquet reaction, forty-eight hours after admission, human negative, bovine negative.

Urine on admission: reaction acid; no albumin; no sugar; no acetone. Microscope shows urates and few blood cells. Blood on admission: leukocytes, 16,300; polymorphonuclears, 57 per cent; lymphocytes, 43 per cent.

December 3, 1914. Position of the head has gradually corrected itself since admission. Much less tenderness on moving the head. Guards the neck very carefully and will not voluntarily move the head. Temperature has been running a little evening rise, otherwise his general condition has remained fairly good.

December 15. Patient moves his head to the right on suggestion, but still prefers to hold it to the left. In relation to possible intestinal origin of the trouble, child placed on a buttermilk diet. Also taking aspirin.

December 22. Child now holds his head without lateral flexion; can turn with fair degree of ease both to right and left. The right trapezius is held rigid. Aspirin and buttermilk diet discontinued because of digestive upset.

December 24. Marked rigidity of the neck posteriorly. No headache; no Kernig sign present. A lumbar puncture was made; under slightly increased pressure, 10 c.c. of clear yellow fluid with a slight greenish tinge was obtained. Cell count, three per field. Lymphocytes, 88 per cent.; globulin reaction (ammonium sulphate) positive. Upon standing two hours, moderate coagulation. No organisms on smear or blood culture. The removal of the fluid seemed to alleviate the retraction of the neck.

December 28. A second lumbar puncture, not under pressure, shows the same distinctly yellow color. Renewed examination of the child shows a slight remaining left torticollis and dilatation of the left pupil; marked apathy, insensible to the prick of a pin; any real anesthesia impossible to determine. Right side of the body, from the face down, shows weakness. Patient unable to raise the leg, and able only with difficulty to lift the arm. Knee-jerks exaggerated; ankle-clonus present.

December 30. Examination by Dr. J. J. Thomas: Cervical sympathetic paralysis on right side; weakness of sternomastoid; contraction of pupil; narrow palpebral opening and dryness of right side of face. Right arm and chest give suggestion of beginning flaccid paralysis; the right leg gives a suggestion of beginning

spastic paralysis with ankle-clonus. Positive Babinski; exaggerated knee-jerks. No definite anesthesia can be made out.

January 2, 1915. Examination by Dr. Harvey Cushing: Face shows peculiar sardonic smile. The right eye shows narrowing of the palpebral fissure, some inophthalmos, and a contracted pupil. Vasodilatation persists on pinching the right cheek. Diagnosis of right sympathetic paralysis, cervical. Both pupils react to light; ocular movements normal; globes parallel. Ophthalmoscopic examination negative on the left side and impossible on the right side because of contracted pupil. Tongue protrudes in the median line. Body shows peculiar insensibility to pin-prick. There does not appear to be any complete anesthesia. Patient unable to move the right leg; only in part the right arm; deep reflexes easily elicited without clonus; perhaps better on right than on left. Babinski sign positive, both right and left. No abdominal reflex obtained. Positive findings: (1) muscular weakness on right side of body; (2) right cervical sympathetic paralysis.

January 11. New Roentgen-rays of the cervical vertebræ show an evident carious development in the bodies of the third and fourth vertebræ, a condition not noted in earlier radiographs. General condition of the patient shows slow downward progress. Head traction applied. Wassermann reaction negative.

January 18. General condition very poor. Lungs seem to show pulmonary edema; numerous moist rales throughout; no definite areas of impairment. Breath sounds exaggerated but not bronchial. Face shows on the right side marked flushing, plainly demarcated at the midline; left side shows pallor. This is evidently due to the paralysis above noted.

January 25. Despite the frequency and persistency in the pulmonary edema the patient's condition remains about the same, and the child has been, for the most part, very comfortable. He is kept in a sitting posture, which seems to afford some relief.

January 28. Condition today the same until 10 P.M., when breathing suddenly ceased.

Discussion. In this case, the two interesting points are the cervical sympathetic paralysis and the results of the examination of the spinal fluid.

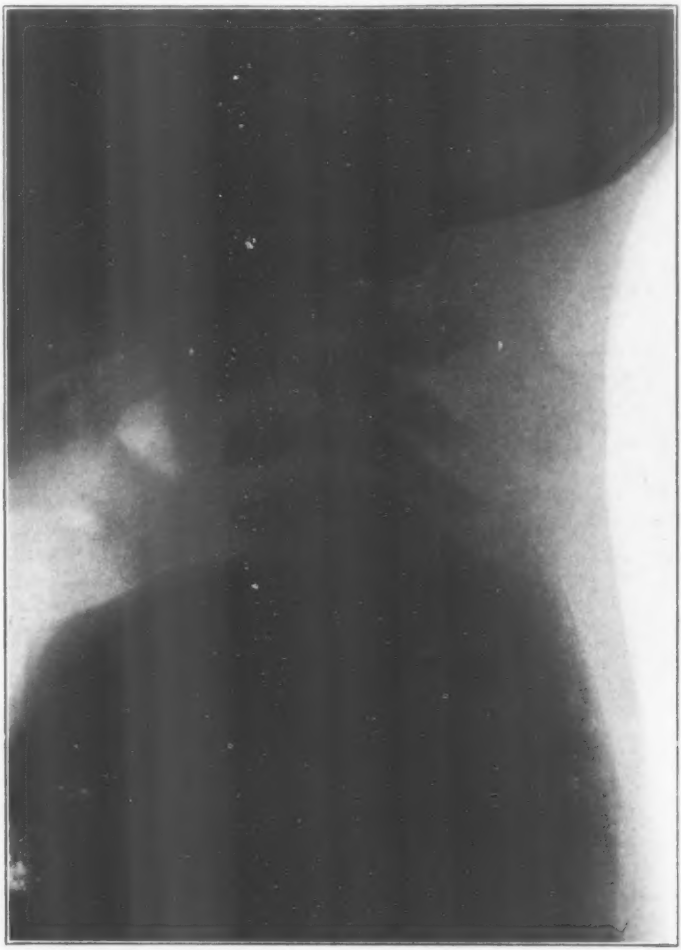
The records of the cases of tuberculosis of the cervical spine admitted to the orthopedic house service of the Children's Hospital from August 1, 1907, to February 1, 1915, reveal the fact that in a total of twenty-three not one shows the paralysis above noted. Of several standard text-books on orthopedic surgery, only that of Tubby[1] mentions the possibility of its occurrence.

This paralysis may result from torticollis *per se*, without changes in the spinal cord.[2] In this case, however, the radiographic demon-

[1] Deformities Including Diseases of Bones.

[2] See Tubby, loc. cit.

stration of lesions in the fourth cervical body supports the assumption of a spinal origin. The syndrome is the result of interference with fibers in the cervical cord running from the ciliospinal centre.



Roentgenogram of the cervical vertebræ, showing caries in the bodies of the second and fourth vertebræ.

A review of the work of W. Mestrezat,[3] in which the characteristics of the spinal fluid in Pott's disease have been well summarized, will lend differential interest to the consideration of the present case.

[3] Le Lequide Cephalo-Rachidien, normal et pathologique, Paris, 1912.

As quoted by Mestrezat, Foi and Sicard found when the disease was at its height the following:

1. "Xanthochromie," or distinct yellow color, quite pronounced.
2. An abundant coagulation, either spontaneous or found after the fluid has stood for several hours.
3. The presence of albumoses.
4. A greatly increased quantity of albumin.
5. No glycogen or, if present, a very small amount.
6. A striking capacity for hemolyzing the red-blood corpuscles of rabbit's blood.
7. The cell changes are nil, or, if any are present, they are slight.

When the disease has not progressed very far, the following findings:

1. Albumin present.
2. Albumoses present sometimes.
3. The yellow color very much diminished or entirely lacking.
4. No cell changes.
5. No hemolytic properties.
6. Glycogen content not diminished.

Sicard claims the above modifications are characteristic. In his cases, of twenty fluids examined ten were colorless and ten had the distinct yellow color; albumin was always present; in five cases the albumoses were found.

Mestrezat, however, questions whether these changes are not the typical changes of the syndrome of "coagulation massive et de xanthrochromie," of which twenty-one cases were collected by him from various sources.

"The syndrome of massive coagulation and xanthrochromie" is shown by typical cases or by so-called "cas frustes," which are variants from the usual type. The two main characteristics of the fluid of this syndrome are marked (quite often rapid) coagulability and the distinctly yellow color. The coagulation at times is so marked that the test-tube may be overturned without spilling any of the fluid.

Of the twenty-one cases collected, the first case was reported by Lepine; later cases by Froin[4] (1903) and Sicard;[5] then Babinski, Cestan and Ravant, Verhagen and Dustin,[6] and others. The primary underlying diseases and conditions were the following: meningomyelitis, tuberculous meningitis of cerebrospinal type, Landry's paralysis, compression of the cord by sarcoma, other neoplasms of the cord, pachymeningitis, and Pott's disease.

[4] Inflammations meningels avec reactions chromatique, fibrineuse, et cytologique du liquide Cephalo-Rachidien, Gaz. d. hôp., 1903, lxxvi, 1003; Syndrome de coagulation massive au cours d'une meningite, 1903, lxxxi, 1587.

[5] Syndrome de coagulation massive, de xanthochromie, et d'hemato-lymphocytose de liquide Cephalo-Rachidien, Gaz. de. hôp., 1908, lxxxi, 1431.

[6] Une sol-distant paralysis de Landry avec syndrome de Froin, Malde Pott cervical, Soc. d. Sci. med. et nat. (Bruxelles), 1909, lxvii, 169.

A resumé of the clinical signs and symptoms of the twenty-one cases is rather difficult to systematize and arrange. However, there is a generally habitual localization of the lesions at the level of the lumbar cord; most often paraplegias, spastic or flaccid, are found combined with sphincter disturbances, sensory disturbances, and sometimes painful subjective phenomena. In short, it is a process meningomyelitic in type, combined with a compression of the lower cord, which may arise from the above-mentioned factors. The symptoms then extend upward, as more of the cord is involved by the pathological changes, and a lumbar cul-de-sac is formed which is shut off from the remainder of the subarachnoid spaces.

Case XVII, reported by Verhagen and Dustin (1909), particularly concerns the subject of this article. Pseudoparalysis of Landry, paralytic and sensory disturbances quite well marked, beginning in the upper extremities, which finally involved the lower. The autopsy showed tuberculous caries of the fourth cervical vertebra making, by means of the pus, a medullary dorsal compression of the cord, and a fibrinous purulent exudate surrounded the whole dorsolumbar portion. There was complete destruction of the lower cervical cord.

Thus this case of tuberculosis of the spine showed a cervical lesion compressing the cord with an exudate involving the whole dorsolumbar portion. This could easily give rise to the two factors which Mestrezat claims are necessary to the production of the typical findings of the spinal fluid of the syndrome, *i. e.*, a closed cavity of the lumbar region and a resulting stasis of the fluid. So by reason of the pathology the symptoms must perforce begin at a higher level and progress downward, being unlike the usual type of case. It seems that in this class the case now being reported should be placed.

A more detailed study of the fluid according to Mestrezat shows:

1. Coagulation immediate and spontaneous or after the fluid has remained standing several hours.
2. Yellow color, "xanthochromie."
3. Increased albumin content.
4. Presence of albumoses.
5. No changes in cell findings.
6. Sugar in variable amounts.
7. Endosmotic non-permeability of iodin and of the nitrates; exosmotic non-permeability of collargol and salicylate of soda.

Pathogenesis of these Abnormalities. Babinski (1903) explained the cause as hemorrhage; Sicard and Descomps (1908) gave the preference to a transudative origin of the fibrin and albumin found. These authors, without affirming the existence of a closed cavity in the lumbar region, insisted upon small meningeal pockets, whose walls played the role of transudation. On the contrary, Mestrezat insists upon two factors: (1) the existence of a

closed cavity producing stasis of the fluid in which accumulated the elements issuing from the blood by way of transudation or hemorrhage; (2) an alteration, infectious or toxic in type, in the walls of the cavity, which so affects the vessels that a transudative process or small microscopic hemorrhages are created.

That the closed cavity is a factor may be shown physiologically by the non-permeability of the fluids to colloidal metals. Normally in ordinary meningitic cases (Mestrezat) colloidal metals injected in the spinal canal disappear rapidly. From the chemical point of view the existence of a cavity is evidenced by the facts that the albumins and fibrogens are increased, the spinal fluid is at a standstill and is not renewed, as a result of which no absorption of these elements takes place. Anatomically, by autopsies in six cases, a closed cavity has been demonstrated.

The "cas frustes" of the same author show deviations from the usual findings. These are due to the fact that the compression of the cord is not so forceful or that stasis of the fluid is not complete. Such cases lack one or more of the usual changes; thus the yellow color may be absent, or cell changes may be present and coagulability may be diminished.

The origin of xanthochromie, or the yellow color of the fluid in which no red-blood cells occur, is explained by Mestrezat as follows:

1. By serogenic means, as from transudative processes.

2. By hemolytic means, hemoglobin from entirely disorganized red-blood cells. Such fluids must be from a hemorrhage at least three days old.

3. By icteroid means, such as occurs in jaundice, when the spinal fluid shows a yellow color.

The diagnostic value of a fluid with a yellow color, therefore, is that it indicates icterus, minute meningeal hemorrhage, at least three days old, meningeal inflammation with intense congestion, or finally a symptom of the syndrome of massive coagulation and xanthochromie. Of course, this excludes the presence of actual hemorrhage into the canal, as evidenced by a yellow color and the discovery of red-blood corpuscles either macroscopically or microscopically, the "erythrochromie" of Mestrezat. This last is sometimes a manifestation of cerebral hemorrhage (Stewart[7]).

According to Nonne[8] (1913) the cause of xanthochromie has not yet been determined. He questions whether it is due to the passage of blood pigments through uninjured bloodvessel walls or is caused solely by many small hemorrhages. Schrützler claims the coloration comes not from blood pigments, but from color derivatives classed under the general group of luteins. The syndrome has been called by the Germans, "Kompressions Syndrome," Kleineberger, Rundfleisch, Erb, etc.

[7] The Diagnosis of Nervous Diseases, London, 1914, chap. XIII.
[8] Deutsch. Ztschr. f. Nervenh., 1913, xlvii, 436.

In relation to the pathogenesis of the xanthochromie it is interesting to note the observations of Babes[9] (1914) in asystoliques (patients afflicted with a condition of the heart such that it is unable to make a complete systole, or a systole sufficient to expel the blood from the ventricles). In the examination of 10 cases, 8 showed a distinct color, 2 a less distinct; none of the patients had icterus, spinal fluids of all were negative to reaction for bile pigments. After examining these patients he then observed about 50 cases, divided into two classes. The first were local affections such as hernias, hemorrhoids, uterine fibromas. The second included more general maladies, as tuberculosis, pneumonia, acute appendicitis, etc. The first class showed no spinal fluids with yellow color, in the second it occurred in 5 cases (2 cases of pneumonia 2 of appendicitis and 1 of tumor albus, none showing any cord lesion, and 3 showing increase in albumin content). He explains the occurrence in the asystoliques as due to stagnation of the blood and to a transudation of the blood pigment through the vessel walls into the spinal fluid. In the second class of cases he thinks it is occasioned by minute capillary hemorrhages or to permeability of the vessels of the choroidal plexus and a consequent hemolysis of the red-blood cells. While these cases are irrelevant to the syndrome under discussion, they are of interest because of the occurrence of the xanthochromie, without the changes in coagulability, in cases showing no cord lesions or any pathological pressure on the cord.

It is my opinion that the case at present reported should be classed under the syndrome on account of the count of three cells to a field, the yellow color, and the presence of albumin, as shown both by the acetic acid and by the nitric acid test, and the slight coagulability. If the objection be raised that the coagulation was not "massive," it can at least be classed as one of the "cas frustes." As additional evidence, there are the Roentgen-ray plates, showing an evident caries of the third and fourth lumbar vertebræ, and the paralysis and sensory disturbances affecting chiefly the right side of the body. No localization pointing to the lumbar region occurs, but it does resemble the case of cervical caries of Verhogen and Dustin. It is to be regretted that no necropsy was permitted. It is a fair assumption, however, that the process in the cervical vertebræ produced a compression of the cord, and as a result the paralysis and sensory disturbances. In addition a closed cavity, a cul-de-sac, occurred and a consequent stasis of the cerebrospinal fluid. In spite of the fact that the Roentgen-rays showed most probably some tuberculous involvement of the lungs, from any

[9] La xanthochromie du liquide cephalo-rachidien, dans d'autres maladies que les hemorragies cerebrales, les affections du nevraxes et l'ictere. Compt. rend. Soc. de biol., 1914, lxxvi, 671; La xanthochromie du liquide cephalo-rachidien chez les asystoliques, Rend. Soc. de biol., 1914, lxxvi, 313.

clinical evidence, this was not apparently the primary cause of death.

In conclusion, it might be well to quote Mestrezat on the value of the spinal fluid in the diagnosis and prognosis of Pott's disease.

"The examination solely of the cerebrospinal fluid will not assure the diagnosis of Pott's disease. It is, however, valuable for showing a meningeal invasion and revealing a compression, which make a lumbar cul-de-sac, a closed cavity, where the cerebrospinal fluid stagnates. The degree of the observed modifications, their persistence or their disappearance in a case of Pott's disease, permits an appreciation of the importance of the lesion and is a means of following its evolution."

My thanks are due to Professor Harvey Cushing for kindly furnishing me the literature on this subject.

SYPHILITIC BURSITIS, WITH REPORT OF A CASE.

By R. W. Scott, M.D.,

RESIDENT PHYSICIAN, CLEVELAND CITY HOSPITAL, CLEVELAND, OHIO.

(From the Medical Clinic of the Cleveland City Hospital and Western Reserve University.)

Syphilitic involvement of the bursæ, either as a secondary or tertiary manifestation, is a rare condition.

The earlier writers, Hunter, Dupuytren, Bonnet, and more especially Crocq,[1] recognized syphilis as affecting bursæ, but the first clear description of the condition occurring in secondary syphilis was published by M. Verneuil[2] in 1873. Later[3] he pointed out that also in tertiary syphilis, various bursæ were sites of gummata.

Moreau[4] reported seven cases of tertiary syphilitic bursitis, which are included in a list of fourteen cases published by Keyes.[5]

Buechler[6] and Trost[7] have reported four cases of early bursal involvement in secondary syphilis. The former also cites three cases of gumma that came under his observation.

A very complete presentation of the subject with a critical review of the literature and report of a case was published by Churchman[8]

[1] Traité des Tumeur Blanches, etc., 1853.
[2] Gaz. Hebd. de Médecine et de Chirurgie, 1873, 2s, x, p. 22.
[3] Loc. cit., 1873.
[4] Thesis to Faculty of Medicine, Paris, August 8, 1873.
[5] Syphilis as Affecting the Bursæ, Amer. Jour. Med. Sci., 1876, lxxi, 349.
[6] Ueber Bursitis luetica, Medicinshe Monatschrift, 1889, vol. I, 393.
[7] Beiträg zu den Erkrankungen der Gelenke und Schleimbeutel im Verlaufe der Syphilis, Wiener med. Wchnschr., 1889, vol. xxxix, p. 545.
[8] Luetic Bursopathy of Verneuil, Amer. Jour. Med. Sci., 1909, cxxxviii, 371.

in 1909. The literature since has remained silent on the subject until recently when Coues[9] reported a case of syphilitic bursitis in a congenital syphilitic.

From a review of the few cases reported in the literature bursal involvement may occur in either congenital or acquired syphilis, primary in the bursæ or secondary by extension from the neighboring bones and joints. Occurring in acquired syphilis the disease may be classified as belonging to either the secondary or tertiary forms.

The former occurs as a transient affection together with the other signs and symptoms of secondary syphilis. It responds readily to specific medication. The latter appears relatively more frequently, and usually manifests itself in the form of a gumma, presenting the same characteristics as gummata elsewhere. It is insidious in its onset and runs an indolent course, terminating in caseation and ulceration if not treated specifically.

Pain appears only when the integument becomes involved, and even then is not a conspicuous feature of the affection. The majority of cases have appeared in the bursæ about the knee, *i. e.*, eight out of twelve cases observed by Keyes.

The following case seems to merit publication not only because of its rarity but also because it is the only case thus far reported in which there has been a pathological examination of the lesion together with a positive Wassermann reaction.

Case.—S. P., female, aged thirty-four years; married; occupation, housework. Admitted to the City Hospital on the service of Dr. E. P. Carter[10] complaining of "swollen knees."

Family History. No bearing on present condition.

Personal History. Always in good health until one year after marriage, at the age of fifteen. At this time she had a diffuse eruption over her body accompanied by sore throat and loss of hair, for which condition she received internal medication for one month. With the exception of enlarged glands of the neck, which were removed by operation ten years ago, the patient has remained well until the onset of the present trouble for which she entered the hospital.

Present Illness. Two years ago the patient noticed a small, hard, round swelling over each knee, which has increased slowly in size to the proportions shown in Figs. 1 and 2. Only during the last few months has she experienced any pain referable to the knees.

Examination showed a hard, indurated, and rather sharply circumscribed painless swelling over the anterior aspect of each knee, about equal size on the two sides. There was no evidence

[9] Luetic Bursopathy of Verneuil, Boston Med. and Surg. Jour., 1915, clxxiii.

[10] The writer is indebted to Dr. E. P. Carter for permission to report this case.

of an acute inflammatory process, and no signs of effusion were present. The tumor was firmly attached to the skin from above and to the patella from below, although it was freely movable over

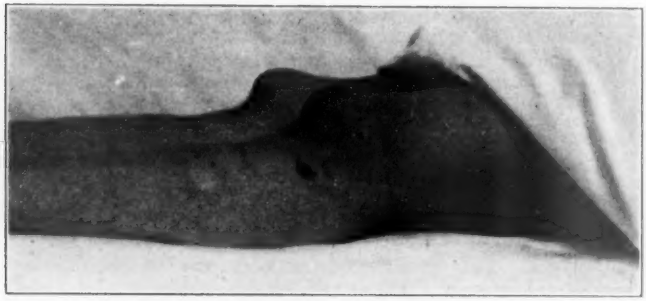


FIG. 1.—Chronic syphilitic prepatellar bursitis of two years' duration. Patient, female, aged thirty-four years. Primary lesion at age of sixteen years. Knees extended.

the knee-joint proper. Flexion of the knees elicited no pain. Circumference of knees over midpatellar region measured 33 cm. Roentgenograms showed no involvement of the bones.

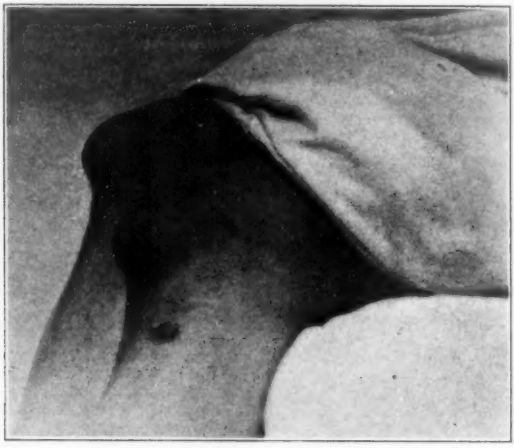


FIG. 2.—Same as Fig. 1. Knees flexed.

With the definite history of a syphilitic infection twelve years ago, the presence of a positive Wassermann reaction in the blood, and the absence of a traumatic factor (housemaid's knee), the syphilitic nature of the swelling was suspected. The tumor on the

left side was removed by operation when it was found to involve the prepatellar bursa and to be adherent below to the patella.

Both microscopic (Fig. 3) and gross examinations showed a typical gumma.

FIG. 3.

The patient was given antisyphilitic treatment for three months at the end of which time the remaining gumma was resolved.

Circumference of knees over midpatellar region now measured 30½ cm.

A CONSIDERATION OF CARDIOSPASM, WITH THE REPORT OF A CASE.

BY B. B. VINCENT LYON, A.B., M.D.,

CHIEF OF CLINIC, GASTRO-INTESTINAL DEPARTMENT JEFFERSON HOSPITAL; PATHOLOGIST TO METHODIST HOSPITAL; ASSISTANT PATHOLOGIST TO GERMAN HOSPITAL, PHILADELPHIA.

CARDIOSPASM, once thought to be a rare disease or condition, is being recognized much oftener on account of our increasing familiarity with its symptoms and the technical measures necessary to its direct diagnosis.

Only slightly more than a decade ago Mikulicz[1] estimated that about 100 cases had been reported. Since then Gottsteim[2] has added 25 additional cases. Bassler[3] speaks of 27 cases that he has treated. Plummer[4] has placed on record 40 cases. Besides these larger series of cases the following additional ones have been published during 1914 by Guisez,[5] Long,[6] Leuiller and Guisez,[7] and Reed,[8] and the subject has been treated by Geppert,[9] Heller,[10] Verger[11] and Eschbaum.[12] Geppert's article constitutes one of the best monographs on the subject.

The etiology of cardiospasm is a varied one. Many causes have been advanced and all are doubtless capable of proof in certain cases. The following causes have been suggested:

1. Primary cardiospasm (Meltzer).
2. Primary esophagitis (Martin).
3. Primary atony of the esophageal musculature (Rosenheim.)
4. Functional disturbance of the innervation of the esophagus, due to paralysis of the vagus causing simultaneous spasm and atony of the musculature of the esophagus (Kraus).
5. Congenital disposition (Fleiner, Zenker, Luschka and Sievers).
6. Kinking at the hiatus esophagei (Plummer).

Bassler[13] has recently published an article in which he puts forward the view that cases exhibiting obstruction at the lower end of the gullet of the type which we have hitherto thought to be cardiospasm is not cardiospasm but rather "a spasm of the esophageal opening of the diaphragm due to contraction of the muscular fibers of the crura . . . which contracts the esophageal opening by drawing the central tendon of the diaphragm against the front of the esophagus or contracts it at the sides."

In support of his view, which is based on a dissection of 5 fresh cadavera and Roentgen-ray observations of 7 cases of cardiospasm, he seems able to prove (1) that "the lower extremity of the esophagus or the cardiac orifice of the stomach have no or only a faintly developed sphincter," and (2) that the stricture is almost always epicardial, usually occurring at a distance of one vertebra above the cardiac orifice of the stomach, which corresponds to the esophageal opening of the diaphragm.

[1] Quoted from H. S. Plummer, Collected Papers of St. Mary's Hospital, Mayo Clinic, Rochester, Minn., 1905–09.
[2] Keen's Surgery.
[3] Diseases of the Stomach and Upper Alimentary Tract, p. 829.
[4] Jour. Am. Med. Assn., August 15, 1908.
[5] Bull. Soc. de pédiat. de Paris, 1914, xvi, 144 to 150.
[6] Buffalo Med. Jour., 1913–14, lxix, 403 to 409.
[7] Jour. de méd. de Paris, 1914, 2 s., xxv, 413.
[8] Lancet-Clinic, Cincinnati, 1914, cxi, 378.
[9] Centralbl. f. d. Grenzgeb. d. Med. u. Chir., Jena, 1914, xviii, 149 to 187.
[10] Mitt. a. d. Grenzgeb. d. Med. u. Chir., Jena, 1913, xxvii, 141 to 149.
[11] Jour. de méd. de Bordeaux, 1914, xliv, 461.
[12] Med. Klin., Berlin, 1914, x, 141.
[13] New York State Jour. Med., New York, 1914, xiv, 9 to 11.

Bassler[14] offers no etiological factors tending to produce this diaphragmatic contraction of the esophageal opening. While his paper throws a new light upon the subject, it would appear to be one concerning proper nomenclature, affecting somewhat the pathology but not altering appreciably the symptomatology or therapy of what we now call cardiospasm.[15]

Under normal conditions we know that food on entering the esophagus requires from seven to ten seconds to pass into the stomach —about one-seventh to one-quarter of this time is required for food to pass down to the cardiac portion of the esophagus, where it remains for several seconds until the cardiac sphincter relaxes and the food is passed into the stomach by the peristaltic contraction of the circular and longitudinal fibers of the esophageal muscles. We have ample proof of this (1) in the Roentgen-ray studies of Kronecker and Meltzer, Cannon and Moser, and others, (2) by the common personal observation of experiencing the sudden aching pain of momentary duration felt back of the sternum in its lower third on swallowing too hot or too cold foods, such as soup, coffee, ice-cream, etc., and (3) by the postmortem evidence, after the ingestion of corrosive poisons, that the greatest amount of erosion in the esophagus occurs at its lower third in the neighborhood of the cardia, where contact with the corrosives has been longest sustained.

While there is this normal delay of foods at the cardia, this varies also with the character of the food, its temperature and chemical concentration, the extremes of all these causing a variable inhibition of the dilating mechanism of the cardia.

Idiopathic or primary cardiospasm occurs frequently, as is seen so often in hysteria and in those of neurotic temperament and tendencies, and is here purely functional; but if of frequent occurrence it permits more and more of the retention of foods, often irritating in character and composition, for too long a time in the neighborhood of the cardia predisposing to the development of esophagitis of varied types, erosions, fissures and ulcerations. Thus a vicious circle is produced, the local inflammation disposing to cardiospasm and the spasm permitting of unduly long retention of foods at the cardia, thereby increasing the esophagitis.

The commonest causes of cardiospasm are *primary cardiospasm* of a purely functional type and constituting a local neurosis, or a manifestation of a general neurosis, and *primary esophagitis;* but when both cardiospasm and esophagitis can be demonstrated simultaneously, it is extremely difficult to decide which is primary and which is secondary.

Of the other etiological factors, congenital disposition may be

[14] Loc. cit.

[15] Heed and Gross in an excellent review of the subject conclude that "cardiospasm is primarily the outcome of a hyperirritability of the vagus." Jour. Am. Med. Assn., January 22, 1916.

an active element in certain isolated cases. Indisputable proof of this, however, should be furnished by the history and the absence of all other etiological factors before one could feel reasonably safe in assigning this as the cause. Likewise, primary atony of the esophagus, while it may rarely occur, is probably also a less common factor in the etiology. Plummer, to whom we are largely indebted for our better understanding of this condition, concludes from a study of his cases that primary atony of the esophageal musculature is a rare occurrence, stating that in his cases "the almost invariable history of spasm at the outset, followed in the later period by the evidence of dilatation—that is, retention of food in the esophagus—is most convincing evidence that the spasm precedes the dilatation and that primary atony is rare." Functional disturbances of the innervation of the esophagus due to the paralysis of the vagus, and kinking at the hiatus esophagei, would also appear to be unusual etiological factors.

Symptoms.—There are undoubtedly many cases of cardiospasm so slight as not to give rise to any subjective symptoms and in patients upon whom the diagnosis can be established only by mechanical means. The first subjective symptom usually volunteered by patients is a sensation of discomfort felt behind and usuallyto the left of the lower end of the sternum. This sensation is variously described as a dull, aching pain, sometimes throbbing, sometimes burning, sometimes cutting or a sense of pressure or weight, as if something had lodged low down in the gullet. These symptoms occur only during the ingestion of food and at first may be of short duration, with periodic remissions, during which the patient is able to eat freely and without dysphagia. During this period the esophageal musculature is sufficiently strong to overcome the spasm and permit of the entrance of food into the stomach with only momentary delay. As the condition progresses compensatory hypertrophy of the musculature must develop to overcome the increasing obstruction, and here a second symptom makes its appearance, namely, the regurgitation of foods from the esophagus into the mouth, very shortly after their ingestion, due to the overactive contracting efforts of the esophageal musculature. The regurgitated foods in this stage may be both liquid and solid, returning in practically the same condition as when eaten and not unpleasant in taste or odor. The majority of the food eaten is passed through the cardia slowly, but nevertheless surely, so long as the hypertrophied muscle proves itself competent to overcome the obstruction. Gradually the muscles tire under their added strain and rupture of the muscle bundles takes place with a resultant dilatation. At this stage the regurgitation of food may be temporarily less frequent and occur at somewhat longer intervals after food ingestion. As the dilatation becomes more extreme the esophageal capacity becomes greater and capable of retention of

larger quantities of food, which are apt to be regurgitated only when the patient is lying down, stooping over, or during a paroxysm of coughing. The dilatation of the esophagus in time may become extreme, with a capacity well over a pint. Since the propulsive power of the esophageal muscle is lacking, food can reach the stomach slowly even when the spasm has been relaxed and in proportion to the weight of the column of food in the esophagus, assisted by gravity. The liquid portion of the meal usually passes more rapidly, seeping through the solid portion so as to leave a dense pultaceous, often foul-smelling mass, usually incorporated with tenacious mucus, which gives rise to a continual sensation of pressure and fulness behind the sternum, with occasional difficulty in breathing, due to pressure on the trachea. As food products are retained for longer and longer periods within the esophagus fermentation and decomposition, chemical and bacterial, take place resulting in secondary esophagitis. At this stage the condition of some patients is truly deplorable; they suffer continually with a sense of burning pressure back of the sternum; they are able to eat only small quantities of food at a time, and their total amount of food ingested and assimilated is so small that they lose weight rapidly; and if not relieved may develop a profound cachexia and die, literally of starvation.

Diagnosis. The symptom-complex is usually so characteristic as to suggest the diagnosis, although I must confess that it is not always so easy as it sounds, for in the case here reported I was at first inclined to believe it one of bronchiectasis. The direct diagnosis can always be made by the use of an esophageal bougie, preferably of the Plummer type, very often by means of the stomach-tube or by fleuroscopic study and the Roentgen-ray plate analysis. In the early stages, with the use of the esophageal bougie or of the stomach-tube, it will be found that an obstruction to the further passage of the instrument in adult cases is met with at about sixteen or seventeen inches from the incisor teeth. The instrument can be passed readily until this point is reached, when an elastic-like obstruction is met with, which under firm and steady pressure usually gives way and permits of the passage of the bougie or the tube into the stomach. During the periodical remissions the obstruction will not be met with, or at times the bougie or tube seems about the pass when it is suddenly gripped in the spasm of the contracting muscle which has been excited by the instrumentation. Great care should be practised in attempting to pass the instrument beyond the obstruction, until the diagnosis of cardiospasm is definitely made to the exclusion of diverticuli, a kinking at the hiatus, stenosis due to malignant disease from within or without the esophagus, or from external pressure by an aneurysmal sac or mediastinal tumor.

To rule out these differential possibilities the use of the Roentgen-

ray diagnosis had best be first employed, which will disclose the presence of a thoracic aneurysm, mediastinal growth, the presence of diverticuli, and the irregular outlines of a carcinoma infiltrating the lower end of the esophagus and causing a stenosis. The use of Bassler's cardioplugger[16] will be of aid in securing better Roentgen-ray plates of esophageal conditions. After aneurysm and mediastinal tumor have been eliminated it is safe to proceed with further instrumentation. If either a diverticulum, a kinking of the hiatus esophagei, or a carcinomatous stenosis is suggested by the Roentgen-ray examination it is best to make use of the esophageal bougie devised by Plummer, which consists of a series of olive tips, which are attached to a stout whalebone staff. The olives are perforated from about their middle to the tip. Six yards of thread are then swallowed by the patient, preferably three yards one afternoon and three yards the following morning, which permits of the thread passing well down into the upper coils of the intestines and becoming fixed so firmly that strong traction can be made on the proximal end emerging from the mouth. The olive tip is then passed over this thread, and by means of traction on the thread the olive tip can be safely guided through the cardia. By varying the amount of traction the sound can be introduced into a diverticulum and its depth and size determined.

The diagnosis of cardiospasm may likewise be confirmed by a test which I believe has hitherto not been reported—namely, by esophageal lavage. With the tip of a stomach-tube in the esophagus at a point just above the obstruction, water is allowed to run in from a graduated glass tank. It will be seen to run much more slowly than if the tube were in the stomach. From 100 to 500 c.c., according to the amount of esophageal dilatation, will run in slowly, but evenly until the flow suddenly stops and the level of fluid in the graduated glass tank begins to oscillate slightly. At this point the water is allowed to escape through the outflow tube, and without changing the position of the stomach-tube it will be seen that the amount recovered is equal to the amount introduced. When the capacity of the esophagus has been reached, if instead of opening the outflow tube the fluid is allowed to remain in the esophagus, by its weight, assisted by gravity, it will cause the cardiospasm to relax. This will occur in a varying number of seconds, according to the degree of the spasm, and will permit some of the water in the esophagus to pass into the stomach, thereby allowing an additional flow from the glass tank, which will then usually proceed in a uniform way until recovered by introducing the tube *farther into the stomach*. While for purposes of demonstration this latter step may be permissible, it is never wise to distend the esophagus to its point of capacity, as it exaggerates

[16] Bassler, Diseases of the Stomach and Upper Alimentary Tract, p. 829.

the already existing atony. The essential point in diagnosis by this method is the ability to recover *from the esophagus itself* an amount of fluid equal, or nearly so, to that introduced. This is not possible of accomplishment in diverticulitis or in stenosis or partial stenosis of the cardia due to pressure from within or without. *It is also of prime importance to determine the presence of a secondary or complicating esophagitis.* This is possible by means of the esophagoscope, but its use is so formidable to the patient that I prefer to make the diagnosis by the examination of esophageal sediments obtained by a method recently published.[17] The photomicrographs used in connection with this paper were made from sediments obtained by this method. It is necessary to determine the extent and kind of this complicating esophagitis because it indicates the proper application of the principles of treatment, *for as long as the inflammatory condition exists so long will the cardiospasm persist,* notwithstanding efforts directed toward the latter to the neglect of the former.

TREATMENT. In the earlier cases of cardiospasm of the primary type, relief may usually be obtained by the administration of antispasmodics, such as belladonna and atropin, pushed to the limit of tolerance; and with due regard to a possible neurotic etiological factor, the regulation of proper hygiene, and the use of hydrotherapy and exercise, preferably in the open air, should be advocated. If these measures do not suffice, esophageal bougies may be used or the spastic cardiac ring may be dilated by means of dilators of the types suggested by Plummer and by Bassler. When there is a concomitant esophagitis, measures should be adopted toward allaying this before proceeding to, or simultaneous with, the treatment of the cardiospasm. Suitable measures are the lavaging of the esophagus with medicated solutions best determined and controlled by the character of the esophageal sediment If the esophageal erosion or ulceration has become secondarily invaded by bacteria one can use germicidal solutions, such as potassium permanganate, silver nitrate, argyrol, etc., until the bacteria have disappeared from the inflammatory desquamation, when blander solutions, such as boric acid or normal salt solution, can be substituted. The use of an autogenous vaccine prepared from cultures grown from the esophageal sediments will facilitate recovery from the severer types of esophagitis. If the esophagitis is sharply localized in the form of ulcerations or erosions, healing medicaments may be directly applied by means of long applicators introduced through a small-bore esophagoscope or through a rubber tube just long enough to reach from the incisor teeth to the lower end of the esophagus. When the condition has progressed to the stage of esophageal dilatation and atony the use of intra-esophageal electricity is indi-

[17] Lyon, AMER. JOUR. MED. SCI., September, 1915, No. 3, ci, 402.

cated, preferably with the sinusoidal current or the faradic current by means of a suitable intragastric electrode. The negative pole should be attached to the electrode within the esophagus and the positive pole to the external electrode in the form of a hand sponge, which is to be carried over the transverse processes from the seventh cervical to the third dorsal vertebræ and over the sternomastoid muscles, particularly the left, to stimulate the vagus. Before turning on the current the patient should drink a small glassful of water to serve as a better conductor of electricity and to prevent burning by direct contact. The duration of each treatment should not exceed ten minutes, and should be given daily in severe cases until improvement is noted. In those cases showing progressive loss of weight due to inanition it is important to arrange the diet in the form of liquids, the total caloric value of which for twenty-four hours should be over 3000 calories. This can be accomplished by the liberal use of milk and cream, olive oil, butter, egg-nog, soft-boiled or raw eggs, and non-stimulating broths.

In the very late cases that come under observation during the extreme stage of starvation weakness it is perhaps better to do a preliminary gastrostomy and feed directly through the stomach and try to build up the strength of the patient before proceeding to the other treatments. Apropos of operative procedure, Bassler[18] suggests that "in intractable cases the approach to the site and cause of the stricture had best be made by the safer abdominal route rather than through the thorax, and that an operation which has to do with the division of the crura, either at their insertions or the bisection of two of the inner portions of both at the back of the gullet or some point in the esophageal opening is worthy of consideration."

It is necessary to observe certain patients over a long period of time. Relapses from primary cardiospasm, properly treated, are comparatively uncommon, but relapses due to exacerbations in any residual esophagitis are more frequent. This will be seen in an examination of Chart II.

Prognosis. Properly treated patients do well, except possibly those instances of cardiospasm secondary to an esophagitis caused by the action of corrosive poison, which are much more difficult to heal and result in contractions due to scar tissue, which, though healed, may still predispose to spasm. It was my privilege to see 2 such cases in Berlin in Paul Cohnheim's clinic which had been treated respectively for five and nine years.

The following case is reported because it illustrates how stubbornly certain cases may persist when the causative factor is an esophagitis, how relapses are frequent and are due to an exacerbation in the esophagitis and that it is necessary that this should be allayed before the vicious circle of cardiospasm and esophagitis can be broken.

[18] Loc. cit.

CASE REPORT.—M. J., aged thirty-two years; occupation, piano stringer. Born in Russia.

Chief Complaint. The patient first came under observation December 18, 1912, with complaint of "stomach trouble," epigastric pain, morning cough, copious expectoration, and loss of weight.

Family History. Negative.

Past Medical History. Always well and strong until three years ago, and the only illness he remembers was typhoid fever at the age of eighteen years.

Social History. The patient came to America seven years ago, has been married five years, and has two children in good health. Does not use tobacco. Uses light wine occasionally. Denies venereal disease.

Present Illness. Beginning about two years ago the patient noticed an uncomfortable feeling of fulness in the high epigastrium with a sense of weight and pressure occurring shortly after his meals, preceded by a slight stabbing pain referred to just above the xiphoid, which would occur irregularly and be associated with the first few mouthfuls of food taken, and would soon pass off to be replaced by a feeling of weight and heaviness. There was no nausea or vomiting but considerable belching, with partial relief and occasionally sour regurgitations. The patient has been steadily losing weight and strength during the past three years. Seven years ago he weighed 205 pounds, and today weighs 144 pounds (subsequently further loss of weight to 128 pounds), and the greater part of this has been lost during the past year.) Two or three months ago he began to be troubled with coughing, and would sometimes spit up a mouthful of very thick, rather foul-smelling sputum, which would seem to suddenly well up in the back of his throat. This had gradually become worse until now, particularly when lying down, and especially when getting out of bed in the morning, he has paroxysmal coughing, which ends with the spitting up of large quantities of thick, tenacious bad-smelling sputum. Of late he has been having night sweats. On some mornings he may spit up as much as a pint and a half of sputum.

Physical Examination. Frail, anemic-looking adult male; looks tired out and ill; skin dry and cold.

Eyes negative; no pupillary alteration. Hair, nose, and mouth are negative. Teeth are in good condition. Gums and conjunctivæ are pale. Glands negative. Breath is slightly offensive. Neck negative. *Chest:* long, narrow thorax, with flaring lower ribs. Supraclavicular fossa increased in depth, but equally. Lungs: Tactile fremitus increased at right apex and extends to third rib. The percussion note is dull over this area and particularly at the right posterior apex, where expiration is prolonged, and occasionally accompanied by rales. No sign of cavity formation. Heart is normal in size and position; second aortic sound slightly accentuated; no murmurs; muscular quality deficient.

Abdomen. Scars from furunculosis; soft; no distention; no rigidity; no masses; some tenderness to deep palpation one inch below the xiphoid; enlarged area of gastric tympany. Liver, spleen, kidneys, appendix, hernial orifices, genitalia are all negative. Deep reflexes sluggish. Pulse 84, rather low volume and tension; regular in rate; no sclerosis of peripheral bloodvessels. Respiratory rate 20. Temperature 99. Blood-pressure 110–80.

Technical Examinations. A stomach-tube passed three hours after a breakfast of scrambled eggs, bread and tea, a cup of coffee, and one glass of water recovered 500 c.c., a dense, tenacious, sour-smelling mass, taking all night to filter to 85 c.c. of turbid filtrate, testing as follows: Free HCl, 20; combined HCl, 25; total acidity 50. Occult blood negative.

In withdrawing the stomach-tube a paroxysm of coughing was induced and was followed by the spitting up of about 200 c.c. of very foul-smelling, purulent-looking material, grayish-white in color and exceedingly tenacious. This material was negative for free HCl.

December 19, 1912. Full dinner last night at 10.30. Now eleven-hour fasting stomach. Stomach-tube shows marked retention of many meat fibers, potato remains, spinach and prune skins. After lavaging the stomach, which required 3000 c.c. to clean, the stomach-tube was slowly withdrawn seven inches, and almost immediately from the end of the tube came about 400 c.c. of foul-smelling, dense tenacious material so mucoid and cohesive as to form a perfect rope, corresponding in size to the lumen of the stomach-tube and which coiled up snake-like in the conical glass receptacle. This material which is identical in appearance with the "sputum," microscopically contains only food rests, desquamated epithelial cells, many leukocytes, and large numbers of bacteria, chiefly streptococci. It is slightly acid in reaction to litmus, but negative for free or combined HCl. This finding, of course, essentially limited the source of this material to the esophagus, but did not differentiate esophageal diverticulitis, stricture, new growth, or cardiospasm. Subsequently it was noticed that at times the stomach-tube, No. 32 French, would pass easily and at other times with considerable difficulty, meeting resistance at a point about 40 cm. from the teeth. The patient was sent to the German Hospital, January 28, 1913, for further study and treatment and remained until June 13, 1913, under the service of Dr. Henry F. Page, to whom I am indebted for the privilege of studying and assisting in the treatment of this patient.

During the time the patient remained in the hospital his pulse-rate varied from 50 to 88. His respiratory-rate varied from 18 to 24 and his temperature from 96° to 100°.

The *sputum* was examined eighteen times for tubercle bacilli with negative results.

Blood counts showed a moderate secondary anemia with hemoglobin 77 per cent. on his lowest reading. There was a moderate leukocytosis reaching 15,850, with normal differential counts. Sputum inoculated into guinea-pigs, subcutaneously and intraperitoneally, as well as into the liver, did not noticeably impair

FIG. 1.—The plate from which this photograph was made was unfortunately broken, and it does not show the extent of the esophageal dilatation, which reached up to the first rib. (February, 1913).

the health of the animals which were macroscopically normal when killed and microscopic examination of tissues showed no evidence of tuberculosis or other infection.

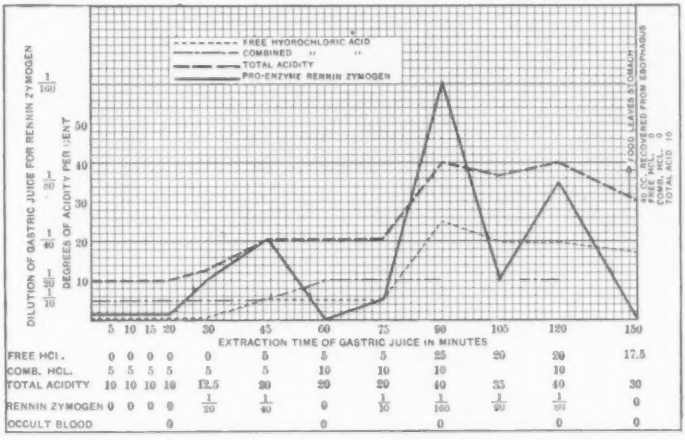
Roentgen-ray examination of lungs shows a very small area at the extreme apex of right lung in which there is marked increase in density. The remainder of both lungs is apparently negative.

Roentgen-ray plate and fleuroscopic examination of stomach made by Dr. A. G. Miller, shows moderate peristalsis; the stomach is moderately dilated, the lower border being just below the

FIG. 2.—Shows the recovery of esophageal tonus. Note the excellent peristaltic wave. (October, 1915.)

umbilicus with the patient standing. The bismuth passes slowly into the stomach in a thin but steady stream, but the majority of it is retained in the esophagus and reaches a point level with the first rib, and the width of the esophagus measures from 1.8 to

4.3 cm., at different levels, being narrowest at the point where the esophagus reaches the diaphragm and widest at a point 18 cm. (7 inches) above the diaphragm (Fig. 1). (*Cf.* with radiographs made twenty-three months later, by Dr. Willis F. Manges, Fig. 2.) There is no evidence of diverticulitis or new growth within or without the esophagus. The Wassermann and Widal reactions were both negative. Esophagoscopy gave unsatisfactory results, nothing further being learned. The patient stood the operation well without the use of anesthesia. Fractional analysis of gastric contents, shows delayed digestion, subacid gastritis and gastric atony. (See Chart I.) Stool examinations were negative.


FREE HYDROCHLORIC ACID
COMBINED " "
TOTAL ACIDITY
PRO-ENZYME RENNIN ZYMOGEN
DILUTION OF GASTRIC JUICE FOR RENNIN ZYMOGEN
DEGREES OF ACIDITY PER CENT
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COMB. HCL. 5 5 5 5 5 5 10 10 10 10
TOTAL ACIDITY 10 10 10 10 12.5 20 20 20 40 35 40 30
RENNIN ZYMOGEN 0 0 0 0 1/20 1/40 0 1/10 1/160 1/80 1/80 0
OCCULT BLOOD 0 0 0 0 0

CHART I.—Fractional gastric analysis of Michael Jurzen. The curve demonstrates delayed digestion, associated with subacid gastritis and moderate gastric atony.

The study of *esophageal sediments* repeatedly showed evidence of esophagitis, with marked desquamation of esophageal epithelium, often fragments of epithelium containing from eight to twenty layers of stratified squamous epithelial cells. Many slides examined showed evidence of ulceration, with areas of necrotic débris with large numbers of pus cells; many streptococci and occasional red-blood corpuscles and all manner of food rests. (See Figs. 3 and 4.) These sediment pictures continued during the first few months of treatment with a gradual subsidence in the inflammatory elements, and at present (August, 1915) the esophageal sediments are negative except for occasional exfoliation of stratified epithelial cells. There is too no microscopic evidence of food rests that were such a constant finding during the early months of observation.

The treatment practised in this case was as follows: (1) lavage

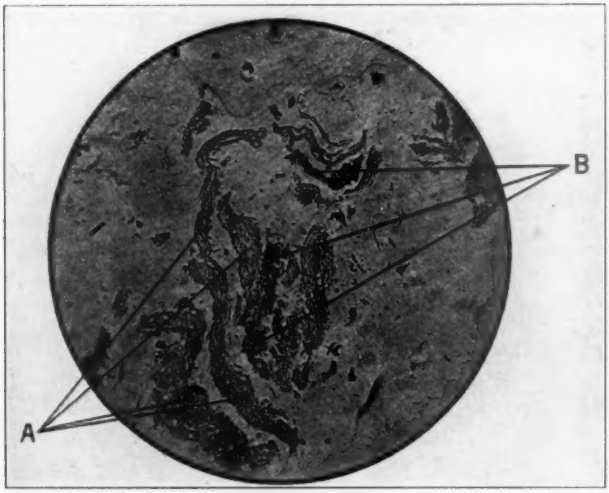


FIG. 3.—Esophageal sediment from a case of cardiospasm. M. J., aged thirty-two years, showing *A*, exfoliated stratified squamous epithelium from esophageal mucosa, with *B*, leukocytic infiltration. × −100.

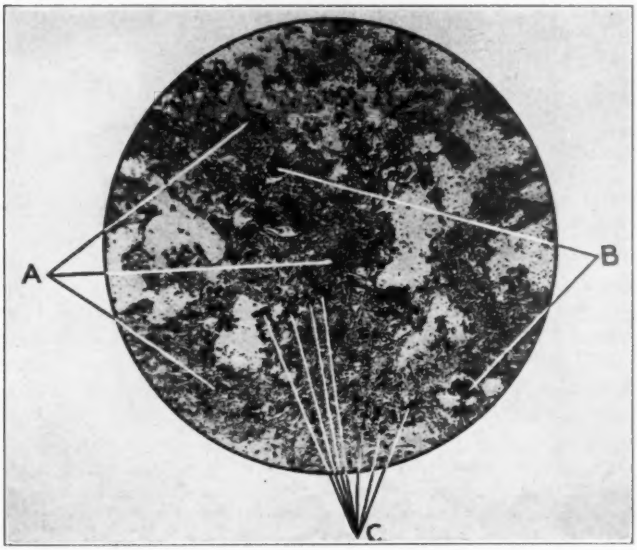


FIG. 4.—Esophageal sediment from a case of cardiospasm, with esophagitis. M. J., aged thirty-two years, showing *A*, exfoliated epithelium containing *B*, masses of streptococci, and *C*, leukocytic infiltration. × −100.

of the stomach with normal saline or various stomachics, such as quassia, condurango and gentian. After which the tube was withdrawn about seven inches, so that its tip rested in the cardiac portion of the esophagus and the esophageal residual contents were recovered either by simple expression or by being gently drawn off by a syringe. For several weeks the amount recovered each day varied between 200 and 500 c.c., and was very thick, tenacious, and very foul-smelling. (See Chart II.) The esophagus was then lavaged,

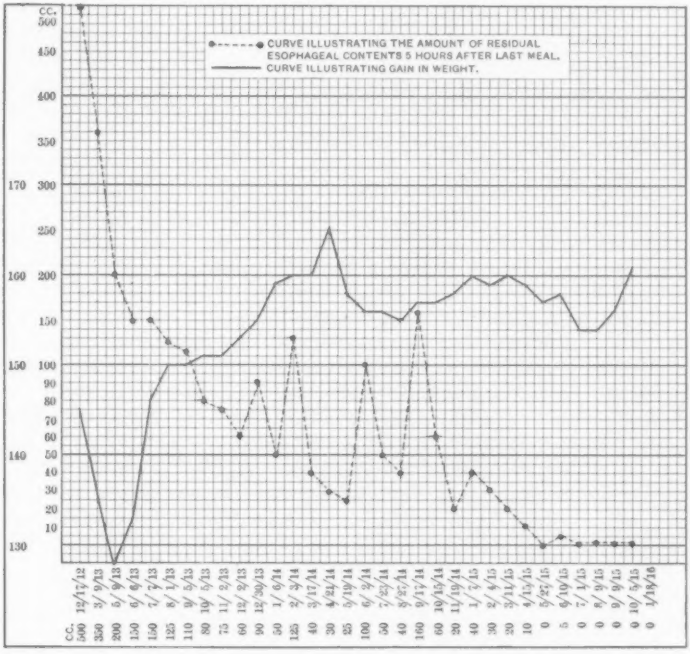


CHART II.—The curves illustrate the decrease in esophageal retention and the increase in body weight as the patient (M. J.) improved.

at first using a solution of potassium permanganate in strength of 1 to 16,000 and 1 to 12,000, until the putrefactive process was under control, and later with solutions of normal salt. During this process of lavaging the esophagus it was found that all of the lavaging fluid could be recovered from the esophagus without having to pass the tube into the stomach. This, I believe, to be pathognomonic of cardiospasm. After lavaging the esophagus, olive-tip bougies, well lubricated, were passed by steady pressure through the spastic cardia, retained in place for several seconds, and then passed

rapidly up and down from four to ten times. At first the tonicity of the spasm was so great as to permit the passage of olive tips, only 5 mm. in diameter, gradually increasing the size to tips 18 mm. in diameter, which can now be passed without effort.

After this an intragastric electrode, properly protected by a perforated hard-rubber tip and a capillary rubber tube, was passed into the esophagus, after the patient had swallowed a glass of water, and the faradic current, and subsequently the sinusoidal current, was used for periods of from five to ten minutes at each treatment. The negative pole being within the esophagus and the positive pole being attached to a small sponge electrode, which was carried over the sternocleidomastoid muscle on either side to stimulate the vagi and over the spinal cord between the seventh cervical and the third dorsal vertebræ. The frequency of treatment was gauged by the amount of the esophageal retention and its character, and by the amount of esophagitis, which was estimated by a study of the esophageal sediments made at frequent intervals. In the first few weeks the amount recovered each day varied between 200 and 500 c.c., and in character was very foul-smelling, and consisted of macroscopic and microscopic food rests, often fermenting, and held tenaciously together by quantities of thick glairy mucus, in which was found microscopically various food rests and many pus cells; stratified squamous epithelial cells from the esophagus, many bacteria, and an occasional red-blood cell. As improvement took place the amount of esophageal retention gradually lessened, and in six weeks had fallen below 150 c.c. Six months later the retention had been reduced to 50 c.c., and gradually became less and less until two months ago (about twenty-two months after treatment was begun), it became impossible to demonstrate esophageal retention of either macroscopic or microscopic food rests.

The *diet* was arranged on a plan of liquid foods of various forms—milk, cream, broths, oatmeal gruel, barley-water, albumen-water, egg-nogs, and olive oil—and was given in small amounts and at hourly intervals, so that the total caloric value for twenty-four hours varied from 3000 to 3500 calories.

Under this plan the patient gained in weight 16 pounds (from 128 to 144) during a period of two months. Gradually his diet was increased to soft and semisolid foods, the latter being gauged by the type of food rests found in the esophageal sediment. His weight is at present 165 pounds, which shows a net gain of 37 pounds. He is on full diet, has no symptoms, and expresses himself as being in better health than he can ever remember, and has been back at his work for about two years.

POLYPOSIS OF THE COLON.[1]

By Horace W. Soper, M.D.,
ST. LOUIS, MISSOURI.

Comparatively few cases of multiple polyposis of the colon are reported in literature. In 1907 Doering[2] collected 50 cases and reported 2 of his own, making a total of 52. In addition to Doering's cases I have been able to collect 8 not heretofore compiled, namely:

Case 1. Dr. P. Y. Tupper.[3] Male, aged ten years. Simple adenomatous polyposis of the rectum and colon. Operation: colostomy; died twenty-eight days after.

Case 2. Esser.[4] Male, aged twenty-nine years. Rectum and colon full of adenomatous polypi. Operation: colostomy at ileocecal valve; recovery.

Case 3. S. Oseki.[5] Female, aged thirty-one years. Polyposis of the colon and carcinoma of the rectum.

Case 4. E. H. Terrell.[6] Multiple adenomata of the rectum; diagnosis by proctoscope and treated locally.

Case 5. H. Lillienthal.[7] Female, aged twenty years. Polyposis of the entire colon; rectum free. Operation June 5, 1900. Resection of the entire colon and ileosigmoidostomy; recovery. Patient presented to New York Academy of Medicine January 14, 1901.

Case 6. Z. von Bokay.[8] Child, aged six years. Multiple benign adenoma of the colon. Died two hours after resection of the colon.

Case 7. E. A. Babler and H. J. Niebruegge.[9] Female, aged nineteen years. Carcinomatous polyposis of the colon. Operation; appendicostomy; died nine days after the operation.

Case 8. W. C. Carroll.[10] Male, aged thirty-eight years. Intestinal polyposis. Operation: cecum, ascending colon, and half of the transverse colon removed for multiple adenoma; recovery.

The following is a summary of the analysis of the grand total of 61 cases:

1. The growths are most frequent in children; the ages, however, range from five years to sixty-two years. Twenty-six cases, or 43 per cent., showed the presence of carcinoma. The most frequent

[1] Read at the meeting of the American Gastro-enterological Association, May 10, 1915.
[2] Arch. f. klin. Chir., Band lxxxiii, S. 194.
[3] Paper read at St. Louis Surgical Club, March, 1913.
[4] Deutsches Archiv. f. klin. Med., 1908, Band xciii, S. 535.
[5] Deutsche Zeitschrift f. Chirurg., Leipsic, 1912, cxviii, No. 5, S. 463.
[6] Proctologist, St. Louis, No. 3, p. 158.
[7] American Medicine, April 27, 1901, p. 164.
[8] Jahrbuch f. Kinderheilk., 1913, Band lxxviii, S. 184.
[9] Jour. Am. Med. Assn., April, 1909, No. 16, p. 1235.
[10] Surg., Gynec. and Obst., No. 4, xx, 412.

site was in the rectum, sigmoid, and splenic flexure. Adenocarcinoma was the type of growth in all the cancer cases.

2. The small intestine is rarely involved; the ileum 5 times, jejunum and duodenum 4 times. A tendency for the growths to occur in members of the same family has been observed, notably in the cases of Doering,[11] Cripps,[12] Paget,[13] Smith,[14] Bickersteth,[15] and Zahlman.[16]

3. In 5 cases of benign adenoma, carcinomatous degeneration was observed in various polypi at different places in the colon. The cases of Handford,[17] Petrow,[18] Forster,[19] Doering,[20] Wulf,[21] Hauser,[22] and Bardenheuer.[23]

4. Esser[24] did a colostomy at the ileocecal region. Patient up and about in fairly good health a year later. W. C. Carroll,[25] in the Mayo Clinic, resected the rectum, ascending colon, and one-half of the transverse colon, with the recovery of the patient. Lindner[26] resected the rectum and descending colon, with recovery; the patient was in good health four years after operation. Lilienthal[27] resected the entire colon, performing an ileosigmoidostomy, with recovery.

The case which I present is therefore the second one on record in which the entire colon was successfully resected for polyposis.

Case Report. Male, aged eight years. Came under observation October 14, 1912. He has had a diarrhea since infancy. Two years ago blood appeared in the stool, followed by anal prolapsus. A polyp the size of a walnut was found on the first valve of Houston, removed by clamp and suture by Dr. Ellis Fischel. A second growth in the mucous membrane of the cheek, pea-size, was also removed. Both showed the same histological structure, namely, simple adenoma. The symptoms of prolapsus did not return, but blood persisted in the feces. December, 1912, two pedunculated polypi, hazelnut size, three inches from the anal margin, were removed through the sigmoidoscopic tube by the snare and chromic acid bead cauterization. In April, 1914, four growths, five inches from the anal margin, hazelnut size, were removed in the same manner. Occult blood persisted in the feces, and suspicion was aroused that

[11] Loc. cit.
[12] Transactions of Pathological Society, London, 1882, xxxiii, 137.
[13] Cited by Doering, Arch. f. klin. Chirurg., Band lxxxiii, S. 194.
[14] St. Bartholomew's Hospital Reports, 1887, xxiii, 63.
[15] Ibid., 1890, xxvi, 122.
[16] Jahrbuch über die Leistungen u. Fortschritte d. ges. Med., 1903, S. 297.
[17] Transactions of Pathological Society, London, 1890, xli, 133.
[18] Abstract in Centralbl. f. Chirurgie, 1896, S. 542.
[19] Rev. Méd. de la Suisse, Rome, 1903, xxiii, 362.
[20] Loc. cit.
[21] Inaug. Dissert., Kiel, 1892. Cited by Doering, Arch. f. klin. Chirurg., Band lxxxiii, S. 194.
[22] Deutsches Archiv f. klin. Med., 1895, lv, S. 429.
[23] Arch. f. klin. Chir., 1890-1, xli, S. 887.
[24] Loc. cit.
[25] Loc. cit.
[26] Centralblatt f. Chirurgie, 1896, S. 935.
[27] Loc. cit.

all the colon might be involved. Abdominal section was determined upon.

Operation, April 18, 1914, by Willard Bartlett. The entire colon was excised. An end-to-end anastomosis was made low in the sigmoid with a Murphy button.

The boy made a rapid recovery. Subsequent sigmoidoscopic examinations showed no recurrence of the growths. His weight prior to the operation was 41 pounds. One year later his weight was 51 pounds. The rectum and a portion of the sigmoid are markedly dilated. At the junction of the ileum with the sigmoid a sort of valve-like fold of mucosa can be seen. The Roentgen-ray examination confirms the sigmoidoscopic diagnosis of dilatation of the rectum and sigmoid.

FIG. 1.—Roentgenogram showing dilatation of rectum and sigmoid.

The small intestine still contained bismuth in the terminal ileum seven and one-half hours after the ingestion of the bismuth meal. He was kept on a diet of soft foods. Raw fruits and vegetables caused several severe attacks of colic, otherwise he has had no inconvenience. The feces are normally formed, one to two movements daily.

The specimen contains eight growths, varying in size from a pea to a small walnut. (See Figs. 2 and 3.)

PATHOLOGICAL REPORT. Benign adenoma. The special characteristic is the large amount of connective-tissue formation with cystic dilatation of the intestinal glands. (Dr. D. L. Harris.)

The rectum and sigmoid are involved in 95 per cent. of all cases of polyposis of the colon. Blood in the feces is the predominant

FIG. 2.—Specimen of colon, showing eight polypi.

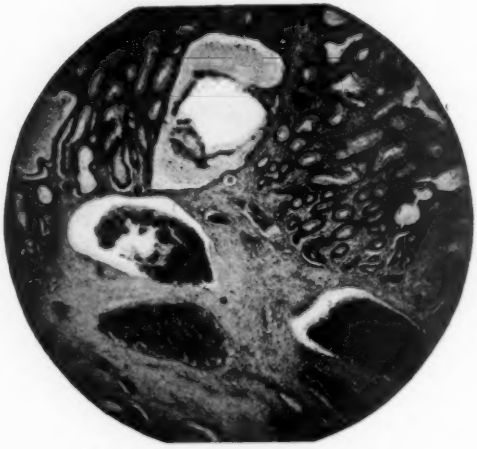


FIG. 3.—Photomicrograph of section.

symptom and should lead to sigmoidoscopic examination, the only means by which an accurate diagnosis can be made. When limited to the rectum and sigmoid the polypi can be readily removed by means of the snare and cautery, provided they are not too numerous; however, the treatment to be advised is complete resection of the entire colon.

MYXEDEMA AND THE NERVOUS SYSTEM.

By Robert L. Pitfield, M.D.,

PHYSICIAN TO ST. TIMOTHY'S HOSPITAL AND TO GERMANTOWN HOSPITAL, PHILADELPHIA.

Most functional nervous diseases, such as many psychoses and neurasthenias, are not idiopathic, but, like many other disease entities, such as anemia, are caused by infections or intoxications, patent or occult. Tuberculosis, auto-intoxications, or defective secretion or functioning of the polyglandular system are responsible for a large proportion of nervous diseases long considered organic.

It is always wise to explore carefully for the cause of such nervous phenomena. Now that we know that latent tuberculosis plays such an important role, every lesion of the disease, no matter how dry, old, and fibrous it may be, or how long standing, always must be reckoned and appropriately dealt with.

Just so every derangement of the thyroid function plays its part in a causative way. In every physical examination the size and consistency of the thyroid should be observed as regularly as the size and functional ability of the heart is noted.

Most individuals with thyrotoxicosis exhibit constantly many of the stigmata of neurasthenia. Quite a distinct form of psychosis (Basedow psychosis) which may precede any objective signs of the disease, is described by Kraepelin, having hallucinations, mistrust and groundless suspicion, dislikes and jealousies, with tremor of voice and hands.

In athyrosis, quite as often, mental disease of various grades depend upon defective thyroid function. In every case there is nerve weakness, trifling or severe.

Melancholia is the form commonly seen, but acute or chronic manias or dementia may have at least as a large etiological factor, athyrosis.

Perhaps of little moment, but deserving of thought, in adults at least, the skin and appendages, the nervous system, the end-organs of special sense, pituitary and the nervous and chromaffin elements of the adrenal glands, all developed from the ectoderm,

are most influenced by this condition of athyrosis. Any weakness of the skeletal muscle and myocardial insufficiency are probably due to lack of tone depending upon nerve weakness. The exhibition of thyroid extract, the most protean of all medicaments, acts in curious and diverse ways. It increases protein metabolism, and oxidization tends to facilitate the mobilization of sugars; if fed to tadpoles it induces the premature change into frogs, while thymus extract, on the contrary, increases the size without influencing morphology.

It seems to be the potent link which binds through the medium of lateral chains, foodstuffs to starving cells, with "Water, water everywhere nor any drop to drink."

Without it nutritive processes cannot definitely end upon tissues, such as nerve cells, designed to receive such benefactions, but are stored up in fatty and myxomatous masses in the skin. As is well known, overdoses produce all the phenomena of Graves's disease.

Generally in the obese it causes a reduction of fat. In a case of incomplete athyrosis, previously reported, it transformed the nutrition of a thin, nervous, almost cachectic young woman by stirring up her metabolism, into a ruddy, plump, healthy woman. In another case the libidio sexualis instead of being increased was oblated, and remained so after the thyroid extract was stopped.

According to Ewald, notwithstanding the wonderful therapeutic result obtained with thyroid extract in myxedema, even if given up to the limit of tolerance, something yet seems to be needed to bring back absolute mental acuity and well-being in these cases.

In no recovered case of this disease have I ever seen any marked mental vigor distinguish the individual.

The manifestations of athyrosis are as protean as in the action of the extract. It is always accompanied by anemia, nervous depression, and weakness. The fatigability of the individual is greatly increased and other neurasthenic symptoms supervene.

If the mind is strong, with no ancestral psycopathic tendencies, the brunt of the disease is not spent upon the brain but upon the other organs. The anemia may be more marked than the nervous atonia, or the disturbances of the sexual organs in woman may meet the deficiency more than any other parts.

In neurology and gynecology chiefly, athyrosis plays the greatest role; but every specialty has to deal with it just as it has to deal with the other great protean diseases, hysteria, syphilis, and tuberculosis.

Severe cases may resemble pernicious anemia in many particulars; even to the blood picture, the asthenia is marked and anemia profound. Cardiac decompensation and glycosuria, as well as the mental and nervous symptoms above noted, may be induced through athyrosis. Extreme cases are commonly mistaken and treated for dropsical nephritis.

In almost all adult cases the following well-known factors are to be found in their histories. Women who have borne children seem to be affected more than any other class of patients; women who are about the climacteric too are affected more than younger or older women. In many cases, as in exophthalmic goitre, a history of severe infectious process earlier in life, with a probable inflammation of the thyroid gland, is noted. Many have had scarlet fever severely; hereditary syphilis as an etiological cause was noted in several of my cases.

The best results in treatment are obtained in women under forty-five years, after fifty years the results are not as good. The following case, an old abscess of the thyroid body with a resulting puckered cicatrix, probably had destroyed enough of the parenchyma of the gland to diminish the functional requirements and cause myxedema in a single woman, aged forty-four years.

Case I.—Miss L., aged forty-four years; incomplete myxedema. Since childhood her sister, an intelligent nurse, said she had been peculiar and hard to understand. When about eight years old, she suffered from a severe attack of scarlet fever and diphtheria, and all her ill health dated from this period. Other than this infection there was nothing in her history of etiological moment.

In entering the room I was impressed with this woman's white, immobile, wooden countenance. The cornea of the white eye was the seat of a large scar, and a glance at her throat revealed a deep puckered cicatrix at the site of the thyroid isthmus. An abscess had formed there during scarlet fever and had been opened. She was very deaf. The various stigmata noted at first glance, *i. e.*, the scars and the deafness, all resulted from scarlet fever above noted.

She was a woman of average height and weight, rather slow and deliberate in her movements, with a high-pitched voice common to deaf people.

Her physical condition was that of an anemic woman past the climactric. Her heart was weak but the lungs were normal. Digestion was good. Menstruation, always regular, began at fourteen years, and had ceased at forty-two years, with hemorrhages at the end. Her blood-pressure was 130; pulse slow and weak. There had been some slight dyspnea, and some edema of the legs and feet. The left kidney was ptosed and painful; pelvic organs were normal, apparently. Her urine was negative. Blood picture: red cells, 4,000,000; hemoglobin, 78 per cent.; polymorphonuclear cells, 62 per cent.; lymphocytes, 36 per cent.; eosinophiles, 2 per cent.

The chief disabilities were nervousness; her station was good; the major reflexes were sluggish. Pupils unequal, due to corneal scar, but responded to light. There was some motor weakness of the legs manifested by early fatigue on standing and giving way of the knees on descending stairs. This is the commonest nervous

symptom in the myxedematous. Occipital headache which was worse on moving her head, also affected her at times.

Her mind exhibited abnormal peculiarities, but hallucinations were absent. She was much depressed, and sat all alone in her home away from her sister and mother. She was jealous of her sisters, and said she believed she was an adopted and not the natural child of her mother. This fixed idea she reiterated again and again to her family. Memory was as good as ever. She felt the cold greatly even in warm weather. She sneezed repeatedly in the sunlight; there was a constant roaring tinnitus aurium.

Her face was myxedematous; the outer half of her eyebrows were thin; her hands were rather clumsy in movements, but delicately formed. Her hair was thin and came out freely; the rest of her skin was normal, save some slight edema of her ankles.

With much difficulty she was persuaded to take thyroid extract. She was told she could not very well get along without it, and having once taken it would procure it herself to relieve depression and other disabilities. More than two grains per diem caused much aching, but on this amount, which she has taken for six months, her depression has disappeared. Her ways are more normal and she is in every way tractable and not despondent. Into her countenance have come lines of expression hitherto obscured by the pad of myxedema. She herself now procures her medicine and depends upon it to keep dispelled the gloom which had so grievously beset her. Her myxedema depended upon a thyroiditis in childhood.

Case II.—Mrs. von L. is the case of a woman verging near to a definite psychosis, with mental depression of a kind that made her life and that of her family miserable. She was in her late thirties, married, and the mother of one child. Weighed 123 pounds, and was five feet, six inches in height. For five years she was afflicted with occipital headaches, great weakness, and very marked anemia, thought to depend upon uterine hemorrhages, which have been most obstinate.

As a child she was healthy and well. Had typhoid fever at seventeen, but no other severe infection. Began to menstruate at twelve, which was always regular and profuse. She was married at twenty-one; child born in one year after, which was premature by two weeks, and was thin and delicate. Has had many induced abortions since.

Five years ago her health markedly failed. She became nervous. A floating kidney was discovered and a rest cure undertaken, without benefit. Her hemorrhages became profuse, and she was curetted and kept in bed unavailingly. She felt the cold bitterly; even in summer she was chilly and uncomfortable. She was generally confined to the house, cried a great deal, and was greatly depressed from no cause at all.

Physical examination showed a plump woman with white, satiny skin. Her face was that of a sad female given to weeping. Dark, purplish circles were under her eyes. Beyond this her face was expressionless and wooden, with blunt lines. Her hair was thin and patches of baldness were seen. Axillary hair absent. Pubic hair very scanty. There were no pads of myxedematous tissues anywhere on her body except her face. Her skin was thin, beautifully smooth. There was no scaling.

There was no swelling of the thyroid body. It could not be felt. Her throat was edematous and voice coarse and harsh. Speech slow, with a tendency to scanning. Her sight was good for near vision, but far vision was poor. Hearing subnormal, with constant tinnitus in both ears; more marked in a deaf ear. Teeth poor. There was pyorrhea. Lungs were normal; liver and spleen not enlarged. Right kidney palpable. There was tenderness over the appendix and epigastrium. She was always obstinately constipated.

Her heart was dilated to the left. Sounds weak; no murmur or thrill noted. Pulse slow and weak. Legs slightly edematous, and she suffered from substernal oppression on exertion.

Blood-pressure, 108 systolic. Examination of the nervous and muscular systems showed nothing abnormal save great weakness, much headache, and depression. She was exacting and unduly suspicious. If people on passing her house at night were heard conversing or laughing she was sure they were talking about her, and she was most unhappy. Any whispering or conversation in a low tone, so that she did not hear, also affected her in the same way. She was unduly suspicious of her husband, and any waiter or servant in a hotel or house was narrowly watched and undeservedly accused of thieving. She was conscious that these ideas were abnormal, but was so depressed that in spite of her efforts she could not dispel them. She never felt well enough to say "good-morning," although she slept overmuch, and could fall asleep at any time during the day.

Blood examination: red cells, 2,200,000; white cells, 5000; hemoglobin, 52.

Anisocytosis extreme. Marked stippling of red cells and other signs of basophilic degeneration. Degenerated red cells are of significance, in this case probably meaning anemia from other causes than hemorrhages. Uterus and appendages normal in size and position; some endometritis.

Her knees gave way under her, and her gait was described as that of a "twitching hop." She could not walk far without exhaustion.

Under thyroid extract, ten grains a day, she gradually became brighter and her depression and headache very much less; is able to walk more and is not suspicious, and is tractable and agreeable;

indeed, is well. At first she suffered from marked decompensation of the heart on taking thyroid extract, but this finally yielded to rest, Nauheim baths, and digitalis.

The atrophic form of myxedema is well illustrated in the following remarkable case, which was so extreme as to resemble the throes of the last stages of pernicious anemia.

CASE III.—Mrs. P. H., Irish nativity, aged forty-one years, was seen with Dr. Walter H. Andrus. When first treated by her physician she was a withered, bedridden hag, resembling an aged, decrepid woman too weak to leave her bed.

Her family history was entirely negative, with no thyropathic tendencies. Her personal history was the usual typical one of myxedema in woman, in that she was in the fourth decade when the disease began, the mother of four children, born in rapid succession. She also had labored hard and worried very much.

In childhood she had suffered from measles and scarlet fever. In the history of many cases it will be noted that almost invariably the subject suffered from scarlet fever in childhood. Her menses appeared first at fifteen years, and were regular and not painful for years. When she married, at twenty-seven, she was a healthy, active-minded woman of 147 pounds. When thirty-five, after four children had been born, a series of illnesses began to sap her strength, and uterine hemorrhages appeared. Her ambition failed, she became weak and languid, and gave up housekeeping. Occipital headaches now appeared, and for eight years she did not remember a day when she was free from them. Her eyelids became swollen at this time, and her face on arising in the morning presented such a grotesque appearance that she was ashamed to go to the door. Coarseness of the skin and features gradually increased; clumsiness in movements, hoarseness in voice, slowness in speech were evident to all. Her headaches were so severe on rotating her head that she was forced to turn her whole body. Her gait became waddling, and she surely fell if she stumbled. If during the day she was unoccupied for ten minutes she would fall asleep in the chair, with her body dropping forward to her knees. At times when on her knees to scrub she was too weak to rise, and her head would sink to the floor. Her skin became dry and scaly, and her hair fell out abundantly. She could not mount a street car without aid. She was dyspneic on the slightest exertion. Her teeth decayed, broke off, and fell out. At thirty-nine years she took to her bed permanently for her final illness, she supposed, suffering from extreme weakness, aggravated by hemorrhages. Her head had to be supported while she drank and ate. Her vision became dim; she could not distinguish objects across the street.

When seen in 1909 she was bedfast, and on this fairly warm April day she was covered with many blankets and comfortables, and was shivering with the cold. Her weight in the nine years of her

illness had declined from 147 to 118 pounds. Her face presented masses of solid edema, translucent in parts; eyelids looked like blisters, and had to be lifted up by the fingers in order that she might see.

The effort to lift up her eyelids caused deep transverse furrows to appear in her forehead. Her countenance was a "mask of sorrowful fatigue and immobility" (Ord).

Her nostrils were broad and swollen and her lips thickened; the lower one was tremendously increased in size. Over the malar eminences was the characteristic hectic-like flush. The rest of her countenance was of a chalk-like pallor. Her old friends under this disguise of disease did not know her.

Her neck was thin and the thyroid could not be seen or palpated. The rings of her trachea could be seen where the isthmus should have been. There were no supraclavicular pads of flesh; her neck vessels pulsated visibly; her head dropped so that her chin touched her chest. Her hands were broad, clumsy and spade-like. Fingers swollen and very stiff, and could be moved with difficulty. Her legs were the seat of a true as well as a solid edema from weakness of her heart. Hands and feet were always cold and often purple.

A large pad of myxedematous tissue covered and enlarged her abdomen, so that her friends supposed her *enceinte*. Over the middle of the right leg there was also a large pad.

The outer half of her eyebrows was most characteristically devoid of hair, as was the axillary and pubic region. Toe and finger nails were markedly incurved. For years there had been an incontinence of saliva, and every morning a wet spot saturated her pillow. Gums soft and spongy; soft palate edematous and hung down to the base of her tongue. Hoarse croaking of voice characterized her speech, which was scanning in character. Frequently she had to stop in the middle of her sentence to clear her throat. In the open air tears constantly coursed down her cheeks.

Her nervous system was depressed. All voluntary movements were retarded; her apprehension was slow; a marked interval elapsed before she could cerebrate sufficiently to answer the simplest question.

She could sit for hours absolutely still in the same position, as if in a trance, oblivious of her surroundings. For recent events her memory was greatly impaired. Indeed, she could hardly carry a sentence through to completion for sheer lack of ability to remember what she had begun to say. Sleep was heavy and stuporous, disturbed by bad dreams and night terrors; she mostly was placid, but at times displayed irritability, and was cranky and exceedingly disagreeable. Hallucinations of sight were frequent; she saw animals and people "flashing about the room." Tinnitus was so persistent and annoying that when addressed she would put her fingers in her ears to stop the roaring.

Her sense of smell was so deranged that the odor of a dead rat constantly annoyed her, and she insisted many times that her family search for a dead rodent when none was present existed. She declared good fresh food was tainted, and with a bad taste, and would not eat it at times.

Constant chilliness and feeling of cold had been present for eight years. She felt as if cold water was being poured down her spine. When seen about the middle of April, a warm day, not requiring an overcoat, she was in bed with two woolen undershirts, a night-gown, a shawl about the shoulders; about her was a blanket, and over her four layers of blankets, a spread, and a down quilt; added to this her bodily heat had to be augmented by hot-water bottles.

A constant occipital headache she declared was the whole trouble, and felt as if a great weight constantly tugged at her and bore her down. When sitting at the table her head was so oppressed by the painful sense of weight that it was bowed to the table.

Paresthesias affected her hands and feet and rendered her fingers clumsy and useless; she could not thread a needle for the past six years because of the delayed sensation, easily demonstrated. She waddled when she walked (hippopotamus gait); for four years she could not dress herself; she could not recognize a button by touch. Her reflexes were delayed, but otherwise normal.

Her vision was misty, probably from vitreous clouding.

Temperature, 97° to $97\frac{3}{10}°$; pulse, 80, small and weak; no arteriosclerosis; blood-pressure under 100 systolic; cardiac dulness extended to right sternal line apex fourth interspace nipple line to the left. There was a double murmur at the mitral orifice; slightly accentuated, pulmonary second sound.

Blood examination: Hemoglobin, 25 per cent.; red cells, 2,030-000; white cells, 3400; polymorphonuclears, 40 per cent.; small, lymphocytes, 40.8; large, lymphocytes, 17; basophiles, 1.5; eosinophiles, 0.7; no nucleated red cells, but many degenerated cells.

Sexual desire lost and intercourse was disgusting. Menses always late and profuse.

Urine 900 c.c. in twenty-four hours; specific gravity, 1010; trace of albumin; no sugar; few cylindroids.

Five days after giving her 2 grains thyroid extract, t.i.d., iron, arsenic and digitalis, her headaches disappeared for the first time in nine years; quilts and half of the blankets were removed, and she felt warm and talked more rapidly. At the end of the week her temperature was 99°, pulse 100 to 110, and her hands began to perspire.

Patient now was quick to understand, and her voice was more normal; the tones were not so harsh; no swelling about the eyes; no furrows in the forehead; face was thinner and edema gone; natural expression returned; she could thread a needle; bowels more regular.

Pulse rose in ten days to 120, temperature 99⅘°. Hands peeling; she could move about. Urine, 1500 c.c. Never slept in the daytime; headaches now gone and felt as if in a dream. No tinnitus; could call downstairs; hearing acute. Itching of skin came on and thyroid diminished. In three weeks patient got out of bed and dressed herself unassisted.

Her thirteen-year-old daughter said that she "never remembered her mother walking and talking like other folks before." After four weeks of treatment with thyroid extract she walked out and was so changed in her appearance that the neighbors did not know her. She speedily resumed her duties in her home as housewife.

After five years she has remained well, under the constant influence of thyroid extract, which has been cruelly denied her through the default of her own gland.

It was as if she had hibernated for eight years and had been incased in some mummifying substance that had benumbed her mind like that of an animal asleep all winter, and had transformed her features into a grotesque mask, and had shiveled her limbs into mere racks whereon to hang her clothes. To add insult to injury, her disease had delicately rouged her prematurely withered cheeks with the bloom of youth. One little vial of a magic medicine such as one reads of in fairy books had transformed the chrysalis into the butterfly and had restored a wife and mother to her family and friends.

Case IV.—Mrs. G., aged forty-six years, had been ill for years with a multitude of ills culminating in a mild psychosis. This was severe enough to convince three physicians as well as her family that she should be committed to an insane asylum, but this was not done from lack of funds.

She had a family history of psycopathic tendencies. Her mother was somewhat deranged after the menopause and died at seventy-four years.

The chief etiological factors in the production of athyrosis were scarlatina in childhood and the birth of eleven children in rapid succession. She had two abortions also. She was prone to much worrying, and labored hard to care for her large family.

Menses began at sixteen, and were uneventful until after forty, when uterine hemorrhages exhausted her, often lasting ten days, and causing her to keep to her bed. Twice her uterus was curetted to cure this condition, but unavailingly.

She was anemic from hemorrhages and dyspneic on even slight exertion. Her heart was atonic and dilated; her legs were dropsical.

She was a large woman with a waddling gait; weak muscles, which kept her confined to home.

Her nervous system was weak and her reflexes sluggish.

Her mental condition was abnormal in that she was melancholic and depressed; she was unreasonable and incoherent on question-

ing; her mind wandered from the subject of conversation, with no power of concentration at all. She was exceedingly voluble and did not finish her sentences; in fact, could not round out a complete sentence at times. She was given to crying, and would weep for days for no cause. She had the usual occipital headache and subnormal temperature.

Pads of myxedematous tissue covered her abdomen and chest. Her face was wooden-like and expressionless.

The abdomen was so large that she resembled a woman late in pregnancy. Her neck was short, wrinkled, and bull-like.

For five years, even on the hottest days, she had not perspired; her hands and feet scaled and dandruff was in her hair.

Her skin had been wrinkled and yellow for ten years, and her finger nails brittle. Saliva drooled from her mouth and wet her pillow at night. Her lips were swollen and livid; her teeth were decayed and the gums bled easily.

For years she was too dispirited and weak to get up for breakfast. She was dyspeptic and obstinately constipated. Her knees gave way under her and she constantly sought a chair to sit upon.

Here is a large, bloated woman at the menopause, afflicted with melancholy and other stigmata of an ill mind, poor in body as well as in spirit, with a decompensated, atonic heart, having hemorrhages and the attendant anemia, ugly to look upon, a care and bother to her family, grotesque, feeble—a mere hulk. She felt that she would surely die, and no doubt wanted to.

Hardly anywhere in medicine are there any such diverse symptoms or groups of symptoms found all dependent upon one causation factor as in athyrosis. Where, for instance, save in syphilis, do we find such widely diverse components of a symptom complex as a diseased brain and heart coupled with uterine hemorrhages and skin lesions? All such apparently irrelevant symptoms are co-related through the medium of thyroid deficiency.

Hertoghe says that the maintenance of the healthy growth of every cell in the body needs the active function of the thyroid gland.

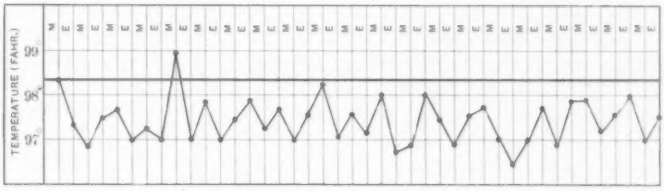
This woman recovered entirely from her mental depression, hemorrhages, weak heart, grotesque appearances, and anemia through the ingestion of thyroid extract, supplemented with iron and arsenic. She, after five years, is now a useful member of her family circle, and is well.

Case V.—Catherine G., aged fifty-three years, married, mother of four children, all since dead, was admitted to the wards of the Germantown Hospital suffering from nervous depression and weakness. She was four feet eleven inches in height, and weighed 200 pounds. She suffered from dyspnea and depression of spirits and body, and was confined to her bed for some time.

She could give no account of her parents, and her personal history was meager and unimportant, save that she had always been healthy until of late years.

She never had had scarlet fever or syphilis; at the menopause she suffered from profuse hemorrhages.

Her intelligence was of a low order. A square, wooden countenance, wrinkled forehead, sparse hair, rough skin, spade-like deformity of the hands, bulky of frame, mental hebetude, and depression rendered the diagnosis fairly easy, but it was complicated by a true arterial and cardiac sclerosis. On admission her blood-pressure was 190 systolic and 100 diastolic. Her heart was slightly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eyebrows were thin; she wept a great deal, and was manifestly unhappy.

Owing to the administration of too much thyroid extract her heart became more dilated, she became more dyspneic and dropsical, and for a while her auricles went into fibrillation. This decompensation of the heart is often noted in many myxedematous cases in the beginning of thyroid medication. For a long time thyroid extract was given without any benefit, even harm, and finally it was stopped and iodides given freely.

No appreciable improvement had been noted in her condition for eight weeks under thyroid treatment, but there was marked improvement under the iodides. A motor aphasia appeared which further depressed her spirits. A new brand of thyroid was secured, and under a daily dosage of 3 grains her depression disappeared; her heart became stronger and assumed its normal size and rhythm. She began to perspire and to feel warm, and discarded her blankets and walked around the ward with a cheerful mien and disposition. She can thread a needle and also feed herself. She still is somewhat dyspneic, weighs 191 pounds, and is myxedematous. Her tinnitus is gone and her aphasia is much better, but is still manifested by hesitancy in speech when she is anxious to talk and excited. The aphasia was probably caused by some arterial degenerative process incidental to her years; it might never have appeared if her brain had not already been predisposed by her athyrosis.

In every branch of medicine do we meet with manifestations of athyrosis even in those not myxedematous, and every man, particularly specialists in the various branches of medicine, should be alive to this condition.

Not only do neurologists meet with it, but obstetricians and gynecologists have to deal with menorrhagias that are not cured by curetments, repeated abortions, sterility, delayed involution of the uterus toxemias of pregnancy and amenorrhea; but surgeons see bones that will not unite in those with athyrosis.

Mysterious anemias, weak hearts, neuralgias, headaches, glycosurias, precardial pain and distress, nocturnal bed-wetting in children puzzle internists, and unless the true cause is known, baffles them.

Some cases of psoriasis and eczema, milk-crust in children, alopecia, and psoriasis seen by dermatologists and are unamenable to treatment unless thyroid extract is employed.

And the same is true in laryngology; laryngitis, aphonia, tinnitus aurium, even deafness, Eustachian stuffing, and intractable catarrhs depend upon athyrosis. Blepharitis in the aged can be cured with thyroid extract.

Not only must extract of thyroid be used, but iron, arsenic and digitalis combined with rest, massage and Nauheim baths must in many cases be employed in order to increase the amperage of the currents of life in individuals thus afflicted.

A convenient laboratory method for the determination of the thyroid content of the blood, just as hemoglobin is estimated, is needed very much in medicine.

To Dr. Walter H. Andrus, of Germantown, Philadelphia, the writer is indebted for notes of two cases and for the privilege of seeing one of them. To Dr. Harry Harvey also the writer owes his indebtedness for notes, etc., in one case seen with him.

FIVE YEARS' EXPERIENCE IN THE TREATMENT OF PULMONARY TUBERCULOSIS BY AN ARTIFICIAL PNEUMOTHORAX.[1]

By Mary E. Lapham, M.D.,

HIGHLANDS CAMP SANATORIUM, HIGHLANDS, N. C.

In 1909 the Highlands Camp Sanatorium began the treatment of pulmonary tuberculosis by an artificial pneumothorax according to the principles established by Forlanini, the author and perfector of the method.

The most important points for us have been the determination of the effect of the method upon the contralateral lesions and the decision as to whether these lesions indicate or contra-indicate the method; the pros and cons of delay; the quantity and frequency of the fillings; the amount of pressure; how best to break up adhesions, stop hemorrhages, prevent and treat pleural effusions.

In determining whether the contralateral lesions permit or forbid an artificial pneumothorax, we have to consider whether the condition of the patient is serious enough to warrant the attempt as an *ultimum refugium;* the extent and the nature of the lesions; the probable effect of suppressing the absorption of toxins; the probable effect of increased intrathoracic pressure upon the better lung and interference with its circulation.

We have had two cases come to us as a last resort, perfectly aware that failure and death were far more probable than recovery; both lived for more than two years, possibly because the absorption of toxins from the bad lung was suppressed. Another case with superficial, fine, wet rales all over the better lung in front from the second to the sixth rib; with absence of first sound of the heart and no palpatory apex beat; soft, irregular pulse and decided tendency to intermission; with the upper lobe of the bad, right lung almost entirely excavated, and the rest of the lung full of degenerating foci, offered little hope of recovery from an illness of more than two years in bed. There was marked cachexia, total loss of appetite,

[1] Read at the Meeting of the Tri-State Medical Association, Charleston, S. C.

periodical attacks of temperature of 101° F. The right lung and cavity was compressed without any difficulty; the rales in the left lung disappeared and the heart action improved. The patient is out of bed, comes up quite a hill for his fillings, and promises a recovery.

Another case of extensive rales in the better left lung with similar cardiac conditions was regarded as hopeless. Again, the compression of the cavities in the bad lung was followed by the disappearance of rales in the other and by improved heart action. This patient is earning his living. Neither the physical signs nor the Roentgen-ray encouraged us to hope for results in these four cases, and it was only as a last chance that they were undertaken.

In the last two cases it is possible that the wet rales were caused by the dilating influence of the toxins, and that this was comparable to an excessive dose of tuberculin upon vessel walls injured in their tonicity by the long-continued action of poisons. The Roentgen-ray showed extensive peribronchitic infiltrations of these left lungs together with old scars, but no unmistakable evidences of activity. In these cases the rales indicated leakage through vessel walls rendered more permeable by tuberculins and not degenerative processes. We have had several bilateral cases similarly benefited, so that the extent of wet sounds in the better lung does not seem to us as prohibitive as it once did, provided the Roentgen-ray suggests absence of active lesions. On the other hand, we have had several cases of absence of rales in the better lung, and apparently only scars revealed by the Roentgen-ray, in which the increased functional demands upon the lung or the increase in intrathoracic pressure caused the appearance of rales in the region of the heart, which in spite of all precautions increased and affected the integrity of the lung. In one of these cases there had never been any abnormal breath sounds or rales in the better lung, and the compression of the bad lung had caused the disappearance of the clinical signs of tuberculosis, when a severe nervous shock brought on an edematous infiltration of the better lung, from which the patient never recovered.

If the contralateral lesions are severe it is possible that delay will increase them so that the attempt can never be made and that prompt action is the one chance. We are often tempted to delay, hoping that the lesions will improve and give us a better chance for holding out against the increased strain. We waited for this improvement recently, but by so doing lost the opportunity. We are not able to predict whether these serious cases will be improved or harmed by delay, and it is only the outcome which tells us whether our judgment has been right or wrong. A case of 103° F. temperature with almost daily chills, aching, and other signs of toxemia improved after resting in bed for a year, so that we were not afraid to undertake the compression of the thoroughly bad lung.

The better lung has steadily improved and the prospects are good. Possibly if we had begun sooner the lung could not have stood it. Several times delay has seemed to assure recovery by getting the lung in much better condition, so that a case which was contra-indicated became possible.

In two cases we should have begun the compression a year before we did. In one there was caseous pneumonia of the right upper lobe which we thought would heal without any interference. During the fall and winter the lung was dry and apparently cicatrizing. There was every indication of satisfactory recovery until the warm weather came, and then rales reappeared and a little blood was coughed up, so that it seemed best to compress a lung that broke down so easily. A good recovery was obtained with working capacity, which persists, but if we had compressed the lung when the patient first came to us we would have saved him nine months' time, all the expense, and loss of working capacity, and the consequent discouragement and dissatisfaction.

A second case was that of a small cavity in the lower part of the right upper lobe, which we were confident would heal. The patient gained in every way for eight months, and then severe hemorrhages induced us to compress the cavity. Our mistake is very evident looking back, but we had just begun the work, and our first principle was never to use the method unless compelled to, and so we cost this patient his time and money and a strong sense of impatience and loss of hope. Another case was wiser than we. Told that compression afforded greatest security against loss of time and toward recovery, he preferred taking the chances incident to dangers and be on the safe side, although the chances were in favor of a prompt recovery without the need of interference.

Murphy, in his unbounded enthusiasm, urges the treatment of early cases regardless of the possibility of recovery without it, and compares the condition to appendicitis with all its danger of delay. Forlanini also advises its use earlier than is commonly deemed advisable.

As we learn the ease and certainty of recovery in uncomplicated cases, it seems more and more as if the risks of delay outweighed those of prompt action. If some public sanatorium would furnish statistics from a large enough number of cases treated before the necessity for the method was apparent, we could judge better whether to still advise delay until sufficient harm was done to warrant our interference, or forestall delay and increase the ease of technique by prompt action. We are in favor of regarding earlier cases as indicating the method and of giving it a much wider field than we have hitherto.

For a time we tried to determine the result of a filling by the area of tympany and the absence of breath sounds, together with metallic clinking or breathing and the coin tests. We find that the

area of the pneumothorax can be roughly mapped out by these means, but that the condition of the lung is not revealed. With all the physical signs of a complete pneumothorax the Roetgen-ray may show the sounder portions of the lung well compressed while dense infiltrations remain prominently exposed and uncompressed. Cavities that give no sign are seen to be surrounded by adhesions, and the use of screen or radiographs furnishes the only reliable information. We try to give as few fillings as possible and to use as little pressure. The complete pneumothorax may easily depress the diaphragm, causing insomnia, loss of weight, extreme nervousness, and rales in the other lung. The pressure may be low, even below that of the atmosphere, and yet the diaphragm be too much depressed. If adhesions hold the heart one way and the gas force it another the action is embarrassed. A weak mediastinal pleura may not register any pressure, but nevertheless, the Roentgen-ray shows it forced over until the other lung or the heart is embarrassed. Deep mediastinal pressure may fatally affect the action of the heart if it is a flabby muscle and the danger not be shown by the manometer. The dyspnea is not necessarily great, nor any other marked discomfort. The distress may be vague and ill defined, and the patient suffer more from inability to sleep than anything else. There is nothing in the physical signs to explain the danger, and only the Roentgen-ray shows it. The readings of the manometer are inaccurate guides when the mediastinal pleura stretches and yields instead of raising the pressure.

The breaking up of adhesions seemingly demands higher pressure, but it is possible that too sudden a rise in pressure may account for many cases of pleural effusions, empyemas, perforations, and spontaneous pneumothorax. In judging of a series of cases like Vollhard's it would be interesting to know the pressure and the Roentgen-ray picture. It is safer to give adhesions time to thin out and not to attempt to stretch them too rapidly. If the thick bands are too resistant, or the synechiæ around cavities are too dense to separate, cavities may have to be compressed by either breaking up the adhesions by pneumolysis or a thoracoplasty tried. Some very discouraging cases of adhesions with uncompressed portions of the lungs containing cavities have finally yielded to low pressure, persistently maintained by the frequent giving of small quantities of nitrogen without causing enough pull on the surface of the lung to cause a pleurisy or perforation. The same is true of cavities with bleeding vessels. In hemorrhage cases it is necessary to watch the compression of the lung with a Roentgen-ray in order to see whether the vessels in the thick walls of a cavity may not be subjected to a strain which will cause hemorrhage when the rise of pressure cannot compress the cavity but simply changes its shape. Although severe cases of progressive hemorrhages demand severe compression, it is not always safe to use high press-

ure, because the cavity may bleed and also because the pressure from below may increase the congestion of the uncompressed portions and favor hemorrhage. Some hemorrhage cases do splendidly for a certain time, and just as all seems to be over and the danger-point passed, a severe hemorrhage may be aspirated into the other lung and cause death. Even a very small cavity surrounded by compressed tissues may torment the patient by the frequent spitting of streaked sputum. Even when there has been dangerous hemorrhages, and we are anxious there shall be no more, high pressure should be avoided unless the Roentgen-ray shows safety. Again, the readings of the manometer may be misleading by reason of the stretching of the mediastinal pleura and a severe hemorrhage first inform us that a deep mediastinal pressure has altered the shape of the cavity and started bleeding. A case of persistent hemorrhages for six years with advanced bilateral lesions, bled so profusely that he was nearly strangled. Half-fainting and all unconscious of our efforts, he was held up from behind in a sitting position while 800 c.c. of oxygen were given. Oxygen was used because he was so livid and lifeless that we were afraid the heart could not stand the strain, and also because we hoped it would have a stimulating effect by being absorbed. The color came back, the patient revived, and all was well until nitrogen was used, because the rapid absorption of the oxygen did not keep up a steady pressure. Eight hundred c.c. of nitrogen were given and an adhesion pulled out the surface of the lung, a tension penumothorax developed, and he died. If oxygen had been used and the pressure raised to +2, the pressure used with the nitrogen, it is quite possible that the increased pressure would have forced a more rapid absorption of oxygen and taken the strain off the adhesion, and there would have been no perforation. We are confident that when the pressure is raised with adhesions, oxygen is safer than nitrogen.

In another hemorrhage case lasting for three days, and frightfully severe, we could get no readings with the manometer. We argued that the lung was stiff and rigid with hemorrhagic infiltrations, and could not be affected by the respiratory movement of the chest wall. We were anxious to prevent any more bleeding by a thorough compression, and considered a Brauer incision, but waited, hoping that the lung would dry out and give respiratory excursions. After two weeks the breath sounds began to be heard faintly over the dull areas, and in a few days more there was no difficulty in obtaining readings on the manometer. The lung was completely compressed without difficulty, and the patient is making a good recovery.

The prevention of pleural effusions depends upon technique and good antisepsis, but even with the best technique it is theoretically impossible, even with a Kjer-Petersen needle, to avoid the carrying in of infection from the deeper layers of the skin. Children coming home from school and filling the house with "colds" or

grippe, or bronchitis, or any other infection, are a good cause of pleurisy from acute infections. Chilling, overphysical strain, overeating, and "bilious" condition, etc., predispose. Infectious pleurisies are common after any acute infection. Static pleural effusions form as the result of too much strain upon the injured vessels; the separation of the pleural surfaces favors the dissemination of tuberculous processes over them. The rupture of a tuberculous focus in the periphery, the forcing of such a focus to the periphery by the collapse of the surrounding tissues; the strain upon adhesions and irritation of the adjacent tissues; the rupture of lung substance and formation of a pulmonary fistula from a cavity to the surface; the pulling out of adhesions and consequent empyemas, are all the result of too much pressure, too hastily applied.

Static and infectious pleurisies demand no treatment beyond that of ordinary pleurisies unless there is too much pressure. As a rule, the pressure rises with an effusion, but occasionally it falls even when the fluid reaches as high as the clavicle. Unless there is some symptomatic indication, rest in bed and the ordinary treatment of a pleurisy suffices; but if the dyspnea becomes marked, or insomnia, or the heart action is wrong, or there are vague sensations of malaise that the patient cannot well define, it is well to remember that an effusion can cause just the same deep mediastinal distress as nitrogen, and that this will not be revealed by the manometer but demands the Roentgen-ray. The effect of the fluid is usually good, sometimes surprisingly so, but there are three conditions that need watching: the expansion of the collapsed lung under the fluid, the penetrating tendency of the fluid and consequent danger of perforation, the conversion of the whole pleural cavity into a cold abscess.

Static pleurisies do not have temperature; infectious pleurisies may have a high temperature and stormy course but they are self-limited; tuberculous pleurisies frequently recover, especially when there is a good eosinophilia; the persistent high temperatures with little remission, with night sweats, and general malaise, suggest a cold abscess which will never be overcome so long as the pleural cavity is a box-like structure. These septic pleurisies can only be overcome by a thoracoplasty.

A pneumolysis may suffice for simply an uncompressed portion of the lung. A cavity held out by adhesions may be thus collapsed; it is possible that a compression of the whole lung may not be needed in order to obtain the obliteration of a circumscribed cavity; when an artificial pneumothorax fails it is quite clearly indicated that the uncompressed upper lobe shall be freed by pneumolysis and compressed. If there is a pleural effusion there is danger of empyema. Cold abscess of the pleural cavity does not seem to be within the reach of a pneumolysis, but Sauerbruch has obtained good results by his method of thoracoplasty. Our five years'

experience of artificial pneumothorax has taught us to question the advisability of allowing patients to go as far as possible before operative interference; it would seem to be a mistake; to prove the best time for undertaking an artificial pneumothorax, some large municipal sanatorium should undertake the study of early cases and report.

We have learned how to induce recovery when there are no complications; to advance the treatment of pulmonary tuberculosis we must go a step farther and learn how to overcome the complications. This demands the coöperation of the surgeon. Surgeons that are distinguished for ability are not familiar with tuberculous conditions or problems. They see the situation entirely from a surgical and not from a tuberculous stand-point; even the most distinguished surgeons enter a *terra nuova* when they begin this work. We need a large sanatorium with an abundance of clinical material, in good surroundings and accessible to the best surgeons, in order that these problems may be worked out for us in the United States as they are being studied in Zurich by Sauerbruch. Our work is behind our needs for lack of sufficient coöperation between the tuberculosis specialist and surgeons of distinguished ability interested in the solving of these problems. There should be a surgical clinic in our large municipal institutions, because these can furnish enough clinical material to give the surgeon sufficient experience. We need a large number of consecutive operations to give us logical inferences. Cincinnati seems ideally situated in this respect. Its municipal sanatorium for tuberculous patients is easily within the reach of the able city surgeons, and its location high upon its breezy hills, affords salubrious surroundings not often found in a city within a short drive. The advancement of our ability to cure tuberculous patients depends upon the surgeons who will take this work up.

AN UNUSUAL MODE OF RUPTURE OF ANEURYSM, EXPLAINED BY DISCOVERY OF ITS DISSECTING NATURE.

By Fred. D. Weidman, M.D.

(From McManes Laboratory of Pathology, University of Pennsylvania, Philadelphia.)

The subject matter of this communication was furnished by an autopsy upon a sailor dying of intracranial hemorrhage from rupture of a saccular aneurysm of the right vertebral artery. The case has been reported from a clinical and morbid anatomical standpoint in another place.[1]

[1] Jour. Am. Med. Assn., 1915, lxv, 1105.

The lesion was of about the size of a pea and lay on the right basilar artery in a large fresh clot. This concealed the aneurysm during the process of severing the cord, during which procedure the aneurysm was either cut or torn in half transversely. Through good fortune the fracture occurred directly at the point of rupture, as was suspected by gross and later confirmed by minute examination.

Grossly it disclosed nothing of its dissecting nature. Its walls were very thin and showed no peripheral fibrosis or adhesions. It contained a small excentric core of fresh clot close to the point of rupture (on the ventral or inferior side) and slightly older peripheral clot with poorly indicated lamination on the opposite side (toward the bulb).

FIG. 1.—Transverse section of entire aneurysm at point of rupture. Magnification ca. 8 diameters. The brackets enclose the vital course of the artery wall. The rest is the aneurysmal sac; *bd*, blood dissection between media and adventitia (cf. with Fig. 2), indicated here by a broad pale channel; *l*, lumen of artery.

Microscopic sections were cut from the lower half of the aneurysm, parallel to the plane of fracture, and therefore in the transverse plane of the aneurysm, the small size of which afforded the unusual opportunity of making histological sections of a whole aneurysm, while good fortune placed the sections at the point of rupture.

The histological report follows: On one side the muscular coat is recognizable as such, vital and divided into three segments by blood extravasations and by a gap. On the other side the wall is

highly attentuated, markedly bulged outward, and encloses a thrombus. The latter does not completely fill the vessel, a broad lumen persisting close to the vital side and communicating with the exterior of the vessel by means of the gap mentioned above. The attentuated (aneurysmal) side of the vessel constitutes fully four-fifths of the circumference. Here the different coats are fused, not recognizable as individuals, hyaline, necrotic, but nowhere ruptured. There are no attempts at organization of the overlying thrombus, which is of very recent type. In the periphery of the thrombus the outlines of the red-blood cells are more or less

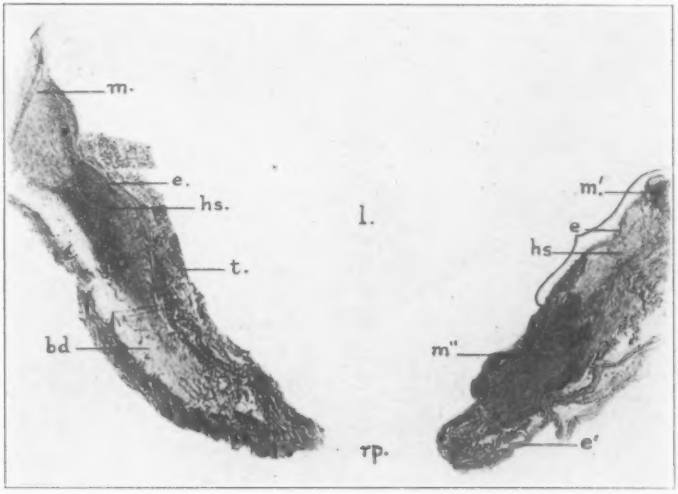

FIG. 2.—Microscopic section showing the artery wall at point of rupture more highly magnified. ca. 50 diameters. *rp*, point of rupture (the "gap" of the text); *m*,*m'*,*m"*, segmented media; *hs*, intersegmental hiatuses containing blood; *e*, internal elastic lamina. It is continued at *e'* through the gap to the outside of the vessel; *t*, slight thrombosis; *bd*, blood dissection (cf. Fig. 1); *l*, lumen of artery. The bracket indicates the position from which a part of Fig. 3 was taken.

preserved and the leukocytes in fairly vital condition, while the luminal part is very recent, showing fibrin reticulum and cellular elements in excellent condition. The point where the dilatation begins is sharply indicated, and here, on either side, the thrombus extends between and separates the media and adventitia, continuing on one side as a narrow zone quite as far as the gap. On the vital side the media is divided into three segments by hemorrhages which show laked red-blood cells and hemosiderin granules. Over the segments the elastica is corrugated, over the defects serpentine or straight, save at the gap, where it is broken. This gap is directly opposite the aneurysmal dilatation. The intima immediately

adjacent is cloddy, granular, and discontinuous, underlaid by laked red-blood cells and no media. It is overlaid by a thin layer of ropy, hyaline, necrotic material suggesting old thrombus. There is no histological evidence of specific causation.

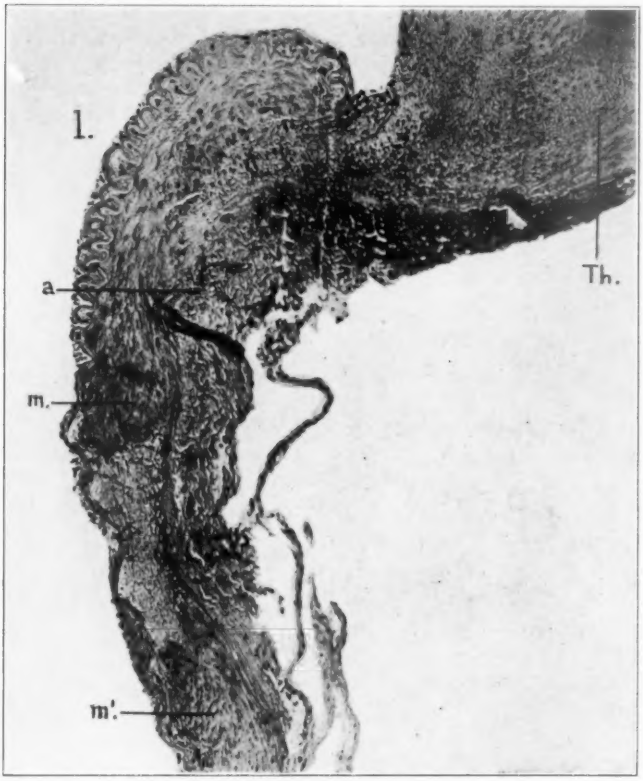

FIG. 3.—Portion of vital part of artery wall near point of rupture (cf. Fig. 2 for location of part and Fig. 1 for relation to whole aneurysm). Magnification ca. 80 diameters. Note corrugated elastica over intact media and stretched elastica over the hiatuses. *Th*, thrombus in aneurysmal sac extending at *a* between media and adventitia; *l*, lumen of artery; *m*,*m'*, segments of media; *h*, intersegmented hiatuses containing blood.

Disregarding gross dissecting aneurysms we find that in practically all saccular aneurysms which rupture the point of such rupture is in some part of the aneurysmal dilatation. Theoretically this is the part which should be weakest, and here we expect, and generally find, the point of rupture if rupture has occurred. In the case here reported we find an exception, the rupture taking place not in

the (apparently) weakest part but through the more vital course of the artery wall.

Study of the microscopic section reveals a probable mechanism, showing that the aneurysm is a dissecting one even though the dissecting features be of but microscopic proportions and extent. Beginning at the thrombus in the aneurysmal sac the hemorrhage may be traced on either side, passing circumferentially (circumferentially in the section—it of course extends longitudinally also in the lesion) between the media and adventitia. This means that blood (and pressure) lay in this position, a pressure which was not a constant one, any more than that in the arterial lumen was, and varying, as there, with systole and diastole. However, on account of the more restricted entrance of blood to this position (it had to pass through crevices) the pressure here was probably not quite equal to, nor the systolic and diastolic variations synchronous with, the luminal pressure. These unequal and irregularly timed forces acting on two opposite sides of a media which is normally accustomed to rhythmical influences are probably responsible for its segmentation, and blood would at once enter the hiatuses, as seen in the sections. After this the internal elastic lamina would be abnormally stretched over the intermuscular hiatuses, lose its capacity to assume a corrugated character during diastole and even be forced outward over the hiatuses during systole. This stretching of the elastica ultimately produced degeneration with consequent roughening and thrombus formation such as is noted close to the gap (Fig. 2). When sufficiently weakened, and unsupported by media, rupture must take place, the adventitia alone remaining to withstand the internal blood-pressure, and shortly also rupturing.

The unusual position of the point of rupture, then, is largely explained by dissective features. This said, it must be acknowledged that the small size of the artery was also contributory. It is most likely that so far as absolute distance is concerned, dissection takes place as extensively in aneurysms of large arteries as in this one. But here, on account of the small size of the vessel, this comparatively short distance sufficed to involve its whole circumference; and on account of the normal thinness of its walls, their coats were separated proportionately greater distances. The degree of disorganization and weaking, then, was much greater in this vessel than would have been the case in an aortic aneurysm, the extent of dissection being the same. The support afforded by the medulla probably prevented an earlier rupture of the major aneurysmal sac.

Against this mechanism it might be objected that the intramural blood appears here as the result of dissection in the opposite direction, *i. e.*, from the point of rupture (not from the major aneurysmal sac), occurring several days prior to death, perhaps from a second miliary aneurysm. That a miliary aneurysm of very short duration

was present, due to the dissecting process, may be admitted. That such occurred as part of the same general influences which produced the major aneurysm cannot be conceded, for we note that only unimportant thrombotic features appear at the gap. With aneurysm there should be notable ones. The internal elastic membrane, too, is degenerated only along a small part of its course, practically only at one point. In aneurysm it should show more diffuse degeneration. And, what is of most importance, the media nearby is not degenerated. It is broken and the point of rupture is a point, not a region. Or it might be objected that the intramural blood entered passively when the aneurysm was severed at autopsy. Against this the histological description makes note of retrogressive changes in the intramural blood (pigment and slight hemolysis), indicating that the blood here is not fresh. It is of some standing.

In conclusion the unusual point of rupture in this case was due to:

1. Support of the wall of the major aneurysmal sac by the medulla.
2. Blood dissection from the aneurysm between the adventitia and media of the non-aneurysmal part of the arterial wall.
3. The normal thinness of this artery wall.
4. Lack of support ventrally, the arterial wall abutting directly upon a serous space.

CALCIUM AND BENCE-JONES PROTEIN EXCRETION IN MULTIPLE MYELOMA.

By N. R. Blatherwick, Ph.D.,
NEW YORK.

(From the Chemical Laboratory of the Montefiore Home and Hospital for Chronic Invalids, New York.)

Since Bence-Jones, in 1847, first observed the presence of a peculiar protein body in the urine of a patient suffering with bone disease, considerable study has been devoted to this subject.[1] Several have investigated the chemical properties of the Bence-Jones protein, but the factors controlling its excretion remain but little understood. Though the present investigation deals primarily with the calcium metabolism of a case of typical multiple myeloma, the output of the Bence-Jones protein has also been followed.

Experimental. The experiment was divided into three intervals, the first of six and the others of three days each. The diet of high protein content was kept as uniform as possible. The patient, who occupied a special room during the experiment, was under continual observation by day and night nurses. The food

[1] Trans. Roy. Soc. London, 1848, i, 55.

was weighed and prepared in a metabolic diet kitchen. The calcium values of the constituents of the diet were calculated from the tables of Sherman.[2] Urines were placed in a refrigerator until the completion of the twenty-four-hour period. Feces, separated by carmin, were dried to constant weight before analyzing. McCrudden's method[3] was used for calcium determinations, that of Folin and Denis[4] for Bence-Jones protein.

Results. In the preliminary period the urinary values for CaO are seen to vary from 0.41 gm. to 0.96 gm., with an average excretion of 0.86 gm. per day; 1.5 gms. were taken in the food while 1.6 gms. were excreted, giving a daily negative balance of 0.1 gm.

Calcium lactate equivalent to 1.18 gms. CaO was administered by mouth each day of the second period. A higher average excretion of urinary calcium (0.84 gm. CaO) resulted. The average daily amount of CaO determined in the feces was 2.67 gms. The balance of CaO with an excretion of 3.51 gms. and an intake of 2.67 gms. was negative, showing a loss of 0.84 gm. daily. Determinations of CaO in the feces of third period could not be made, as bismuth which interfered with the calcium estimations was administered to the patient through an oversight.

The excretion of the Bence-Jones protein was found to be very variable, extremes ranging from 7 to 27.56 gms. Average values for the three periods, 18.56, 17.35, and 19.24 gms. were, however, quite uniform. An increased daily excretion of 2.88 gms. nitrogen in the feces of the second period, followed by 1.81 gms. in the third, may be due to disturbances of absorption, as shown by the development of diarrhea in period II.

Discussion. The plane of calcium metabolism was found to be on a high level. With a daily intake of 1.5 gms. CaO an approximate calcium equilibrium was reached. The diet was purposely chosen with a high content of calcium, as analyses of urine and feces made three months previously had shown an abnormally increased excretion of this element. At that time the patient on his fourth day of uniform diet (1.07 gms. CaO) excreted calcium corresponding to 1.04 gms. CaO in the urine and 1.14 gms. in the feces, therefore failing to retain 1.11 gms. daily. The urinary nitrogen elimination was 16 gms.

In the present experiment, when 1.18 gms. CaO in the form of calcium lactate were added to the daily food, there resulted a loss of 0.84 gm. CaO to the body. Thirty-six per cent. of the calcium oxide administered was utilized, as calculated from the balances of the first and second periods. There was an average daily increase of about 0.1 gm. calcium estimated as oxide in the urine over period

[2] Chemistry of Food and Nutrition, 1911, Macmillan, New York.
[3] Jour. Biol. Chem., 1911–12, x, 187.
[4] Ibid., 1914, xviii, 277.

I, but by far the greater part was eliminated in the feces, which could be expected.

As far as the author has been able to determine, calcium analyses in the excretions of multiple myeloma patients have hitherto been undertaken but twice only. In one instance Seegelken found values which varied inconsiderably from the normal.[5] Williams on two days found 0.92 gm. and 1 gm. calcium, calculated as oxide, in the urine of his patient.[6] The latter undoubtedly represent high values.

The daily amount of this element required by a normal man is placed by Albu and Neuberg at 1 gm. to 1.5 gms. CaO.[7] There are examples on record, however, in which calcium equilibrium was attained on a much lower level, 0.4 gm. CaO daily (Bertram). The per diem elimination in the urine is normally between 0.1 and 0.5 gm., calculated as oxide. Our results, as well as those of Williams, greatly exceed the higher of these normal limits. We were unable to secure a positive calcium balance even when 1.5 gms. CaO was administered. This indicates that the calcium metabolism in our patient was operating at a higher plane than normal. That absorption of calcium was active is shown by the partial retention of added calcium in the form of calcium lactate. *These results justify the view that as a result of the osseous lesions a liberation of calcium has taken place which is shown by an increased excretion.*

Bender found deposits of calcium in the lung of a case of multiple myeloma which came to autopsy.[8] In a second case this investigator reported an increased calcium content of the lungs, lymph glands kidneys, gastric mucosa, and liver. The deposits occurred chiefly in connective tissues. Some years later Tschistovitsch and Kolesnikowa[9] reported a very noticeable increase in the calcium content of the lungs, while smaller amounts of this element were found in the stomach, kidneys, and intestines. These data make it probable that the disease of multiple myeloma, with its attendant destruction of the bony structures, leads to a liberation of calcium which is chiefly excreted, but which in part may be abnormally deposited in certain organs.

The origin of the Bence-Jones protein has always remained a matter of conjecture. Voit and Salvendi inclined to the belief that *less* of the protein *was excreted* on a protein sparing diet.[10] Allard and Weber, who made the first protein metabolism studies in regard to this problem, found no relationship between the amounts of protein catabolized and the Bence-Jones protein eliminated.[11]

[5] Deutsch. Arch. f. klin. Med., 1897, lviii, 276, 286.

[6] Biochem. Jour., 1910–1911, v, 225.

[7] For a discussion of calcium metabolism, see Albu-Neuberg: Physiologie und Pathologie des Mineralstoffwechsels, Berlin, 1906, p. 111.

[8] Deutsch. Ztschr. f. Chir., 1902, lxiii, 370.

[9] Virchows Arch., cxcvii, 112.

[10] München. med. Wchnschr., 1904, li, 1281.

[11] Deutsch. med. Wchnschr., 1906, xxxii, 1251.

Later, Hopkins and Savory, who carried out very careful metabolic studies on three cases of multiple myeloma, came to the same conclusion.[12] Recently, Folin and Denis decided from a study of their case that "there is nothing like a definite or constant relationship between the total nitrogen and the albumose of the urine."[13] The results of our experiments also show a great variation in the amount of Bence-Jones protein eliminated, with no obvious relationship to the nitrogen excretion.

PROTOCOL I.—URINARY EXAMINATION.

Day.	Volume.	Nitrogen.	Bence-Jones Protein.	CaO.
	c.c.	gm.	gm.	gm.
1	1030	13.99	.....	0.4128
2	805	13.15	8.05	0.7511
3	915	13.27	13.73	0.9516
5[14]	1590	17.09	24.91	0.9596
6	945	10.68	27.56	0.6067
7	1030	12.08	19.31	0.8127
8	860	13.00	18.71	0.9761
9	780	10.96	14.04	0.7215
10	1400	13.90	7.00	0.7910
11	1350	11.62	25.70	0.5611
12	1270	13.25	25.02	0.7328

PROTOCOL II.—DAILY AVERAGES OF EXPERIMENTAL PERIODS.

Period.	Urine.		Feces.			CaO balance.			
	Nitrogen.	Bence-Jones protein.	CaO.	Nitrogen.	CaO.	Ingested.	Excreted.	Difference.	
	gm.	gm.	gm.	gm.	gm.	gm.	gm.	gm.	
I.	13.64	18.56	0.7364	0.94	0.8604	1.5034	1.5968	0.0934	
II.	12.01	17.35	0.8368	2.88	2.6745	2.6682	3.5113	0.8431	6 gm. calcium lactate = 1.1766 gm. CaO daily per os.
III.	12.92	19.24	0.6950	1.81					

ABSTRACT OF CLINICAL HISTORY. I. G., male, aged forty-three years, expressman.

Family History. Negative. Typhoid at twelve years; no other previous disease. Admitted January 19, 1915; died July 3, 1915.

Present Iliness commenced two years previous to admission. Persistent pains in chest, back, groins, and thigh; weakness and diarrhea.

[12] Jour. Physiol., 1911, xlii, 189.

[13] Loc. cit.

[14] Part of the urine was lost on the fourth day. It was accordingly not examined. The specific gravity of the urine was normal throughout.

Examination. Thorax shaped like a toad's body, this condition probably resulting from posture and gravity. Exquisite tenderness over the ribs, pelvis, and long bones. No distinct evidence of other organic disease except a diffuse infiltration of the lungs. Roentgen-ray plates showed advanced bone absorption of general nature, including metacarpals and phalanges and a diffuse infiltration (calcium salts?) in the lungs. Blood count: red-blood cells, 3,680,000; white-blood cells, 9000; lymphocytes, 56 per cent.

Deformities of chest increased and severe pains continued until death from pulmonary edema intervened. Diagnosis: multiple myeloma. No autopsy could be performed.

ERRATUM: The diagram which appeared in the article entitled "Some Aspects of the Clinical Study of the Respiration: The Significance of Alveolar Air Analyses," published in this JOURNAL, February, 1916, p. 188, should be corrected so as to correspond to the new diagram herewith shown.

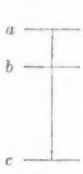

FIG. 1

FIG. 2

REVIEWS

DISEASES OF THE DIGESTIVE ORGANS. WITH SPECIAL REFERENCE TO THEIR DIAGNOSIS AND TREATMENT. By CHARLES D. AARON, ScD., M.D., Professor of Gastro-enterology in the Detroit College of Medicine and Surgery; Consulting Gastro-enterologist to Harper Hospital. Pp. 790; 154 engravings; 48 roentgenograms; 8 colored plates. Philadelphia and New York: Lea & Febiger, 1915.

WITHIN recent years an increasing number of books have been written on the gastro-intestinal tract. Many of them have justified their production while others have seemed somewhat superfluous. In the volume under consideration the author has handled a vast amount of material in such a way as to present the subject matter with great clearness. He has not by any means exhausted his field, but that would be impossible, within the limited scope of such a work. Many notable achievements have recently crowded in upon this branch of medicine covered by Dr. Aaron. Modern research; improved facilities for observation; the development of roentgenographic technic and the perfection of the various tests and reactions have added immensely to possibilities in these lines.

The various chapters are arranged so as to accord with the physiological path of the digestive tract; beginning with oral diseases and proceeding to a consideration of those of the pharynx, esophagus, stomach, liver, gall-bladder, bile ducts, pancreas, small intestines, appendix, colon, sigmoid flexure, rectum, and anus. Throughout these chapters the author must be congratulated upon his endeavor to emphasize the intimate relationship that exists between gastro-enterology and all branches of internal medicine. The author's viewpoint is distinctly that of the internist, although due consideration is given to indications for surgical intervention. In appropriate parts of the book are to be found discussions on the subject of internal secretions; various tests and reactions; qualitative and quantitative analyses for disclosing the condition of intestinal functions; a noteworthy chapter on the examination of the feces; test-meal technic and findings; dietetics; a most valuable chapter on mineral-water therapy; hydrotherapy; mechanical therapeutic agencies; oral sepsis; duodenal feeding and very useful chapters on diseases of the liver and pancreas. A chapter of twenty-six

Examination. Thorax shaped like a toad's body, this condition probably resulting from posture and gravity. Exquisite tenderness over the ribs, pelvis, and long bones. No distinct evidence of other organic disease except a diffuse infiltration of the lungs. Roentgen-ray plates showed advanced bone absorption of general nature, including metacarpals and phalanges and a diffuse infiltration (calcium salts?) in the lungs. Blood count: red-blood cells, 3,680,000; white-blood cells, 9000; lymphocytes, 56 per cent.

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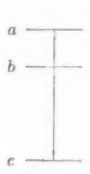

FIG. 1

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REVIEWS

DISEASES OF THE DIGESTIVE ORGANS. WITH SPECIAL REFERENCE TO THEIR DIAGNOSIS AND TREATMENT. By CHARLES D. AARON, ScD., M.D., Professor of Gastro-enterology in the Detroit College of Medicine and Surgery; Consulting Gastro-enterologist to Harper Hospital. Pp. 790; 154 engravings; 48 roentgenograms; 8 colored plates. Philadelphia and New York: Lea & Febiger, 1915.

WITHIN recent years an increasing number of books have been written on the gastro-intestinal tract. Many of them have justified their production while others have seemed somewhat superfluous. In the volume under consideration the author has handled a vast amount of material in such a way as to present the subject matter with great clearness. He has not by any means exhausted his field, but that would be impossible, within the limited scope of such a work. Many notable achievements have recently crowded in upon this branch of medicine covered by Dr. Aaron. Modern research; improved facilities for observation; the development of roentgenographic technic and the perfection of the various tests and reactions have added immensely to possibilities in these lines.

The various chapters are arranged so as to accord with the physiological path of the digestive tract; beginning with oral diseases and proceeding to a consideration of those of the pharynx, esophagus, stomach, liver, gall-bladder, bile ducts, pancreas, small intestines, appendix, colon, sigmoid flexure, rectum, and anus. Throughout these chapters the author must be congratulated upon his endeavor to emphasize the intimate relationship that exists between gastro-enterology and all branches of internal medicine. The author's viewpoint is distinctly that of the internist, although due consideration is given to indications for surgical intervention. In appropriate parts of the book are to be found discussions on the subject of internal secretions; various tests and reactions; qualitative and quantitative analyses for disclosing the condition of intestinal functions; a noteworthy chapter on the examination of the feces; test-meal technic and findings; dietetics; a most valuable chapter on mineral-water therapy; hydrotherapy; mechanical therapeutic agencies; oral sepsis; duodenal feeding and very useful chapters on diseases of the liver and pancreas. A chapter of twenty-six

pages is devoted to roentgenography of the esophagus, stomach, and intestines. Included in this chapter are eleven excellent plates illustrating the various pathological possibilities in the gastro-intestinal tract. The Roentgen ray undoubtedly revolutionized the work of the gastro-enterologist and has brought an element of exactness into his diagnoses which often makes an exploratory laparotomy unnecessary.

One of the tests of any work is the realization or non-realization of the author's aim as set forth in the preface. The author, as he proposed to do, "has put before the practitioner in an orderly, consecutive manner the diagnosis and treatment of digestive diseases" and has made "available all the resources of this branch of medicine."

The book is well constructed; the illustrations are adequate and clear cut. The author and publishers are to be congratulated on a book that should be a splendid working manual for the student and an excellent reference volume for the practitioner.

T. G. S.

MATERIA MEDICA AND THERAPEUTICS. A TEXT-BOOK FOR NURSES. By LINETTE A. PARKER, B.Sc., R. N., Instructor in Nursing and Health, Teachers' College, Columbia University. Pp. 311; 29 illustrations; 3 plates. Philadelphia and New York: Lea & Febiger, 1915.

THE amount of knowledge pertaining to materia medica essential for most efficient nursing is always a debated point. There are those who insist that we are overeducating nurses in this respect and again there are others who feel that there can be no limit to the things we teach those who care for the sick. To either of these, this excellently constructed volume can offer no offense, for it appears to be a happy medium in this respect.

In the preliminary sections, tables, technic, and necessary definitions are clearly stated and explained. The consideration of drugs is arranged by systems; nervous, muscular, circulatory, etc., with an additional section devoted to specifics and drugs which affect nutrition. An excellent chapter on legislation, concerning poisons and habit-forming drugs includes a consideration of the Harrison Drug Act and indicates just what drugs are restricted and how to conform to the law. Chapters dealing with psycho, hydro, electro, serum, and ray therapy are well done and give the reader an accurate idea of these useful adjuncts in the treatment of diseases.

The author has an interesting style and a faculty for clear expression. Only the essentials are presented. Emphasis is not placed upon the fact that a certain drug is prescribed in a certain condi-

tion, but upon what action the drug may be expected to have and what untoward effects may be looked for and the emergency procedure pending the physician's arrival in case of an overdose.

The nurse in training who carefully studies this volume and is permitted to have it supplemented by class-room demonstrations of the appearance and characteristics of drugs along the line indicated by the author will bring to the performance of her duties just that degree of knowledge that is most likely to serve the needs of the physician in attendance and the best interests of the patient.

It is seldom that we can commend a work as heartily as we do this one. The engravings are excellent. The volume is well and carefully indexed. T. G. S.

A Text-book of the Diseases of the Nose and Throat. By Jonathan Wright, M.D., Director of the Department of the Laboratories, New York Post-Graduate Medical School and Hospital, and Harmon Smith, M.D., Surgeon to the Throat Department of the Manhattan Eye, Ear, Nose, and Throat Hospital; Clinical Professor of Laryngology and Rhinology, Cornell University Medical School. 373 illustrations; plates 14. Philadelphia and New York: Lea & Febiger.

This product of the brains and life-work of Jonathan Wright and Harmon Smith impresses the reviewer as being the best work on the subject that America has produced. And this for several reasons: In the first place there is present a wealth of real pathology—pathology that is authoritative, first-hand, and thoroughly digested. This portion of the book could be lifted out and published separately as a splendid epitome of the pathology of the diseases of the upper air passages. In the second place, etiology and therapeutics are correlated with the pathology to a unique and most refreshing degree. And finally, there is running through the entire volume an attractive and very obvious and insistent philosophical note—a note which lifts this book to a plane a bit higher than any publication along similar lines with which the reviewer is familiar. With all of this there is no loss of or neglect of the practical side: no helpful little detail which has occurred to the authors being omitted, no matter how trivial.

Much to be commended is the omission of a section on diseases of the ear. Seldom, if ever, does a single text-book deal with both the nose and throat and the ear and do justice to both. One unpleasant result always follows, and that is a volume of unwieldy size. The day soon should come when, on account of the size of the subject, good text-books on the entire field of rhinology and laryngology can not be written. This tendency is plainly manifest

now, when volumes are being issued on such subtopics as bronchoscopy, the sinuses, the tonsils, etc. But in spite of the increasing breadth of the subject, Jonathan Wright and Harmon Smith have given us a book which covers the entire field, yet withal in so concise a manner as to make a volume of handy size, and yet in so direct and complete a fashion as to be of great help to the mature specialist and a veritable *vade mecum* to the general practitioner and to the beginner in laryngology and rhinology.

The book is well printed, well illustrated, and well indexed. Errata there are and omissions there are, as in all books of this character, but these do not alter the general proposition that rhinological literature has received a valuable addition: valuable not only because it is a unique and authoritative combination of the clinical and pathological, but also on account of the rare philosophical tone which characterizes it throughout. G. F.

MEDICAL AND SANITARY INSPECTION OF SCHOOLS. By S. W. NEWMAYER, A.B., M.D., in charge of Division of Child Hygiene, Bureau of Health, Philadelphia. Pp. 311; 85 illustrations. Philadelphia and New York: Lea & Febiger.

THIS is a good book, and comprehensively written by one evidently familiar with the subject. The reading matter is divided into four parts: administration; school buildings and grounds; infectious, contagious, and communicable diseases; physical defects.

The section on administration includes the organization of bureaus of child hygiene, the duties of the medical inspector and nurse, and various systems of medical inspection. The descriptive plan is comprehensive, and no one can read it without a very good working knowledge of medical inspection. The official statistics taken from the New York reports are good, but those from Philadelphia unfortunately are rather limited and obsolete, being for the year 1910. The medical provisions of the new Pennsylvania School Code are, however, given fully, and serve well as a model for action in other States.

The section on school grounds and buildings is well written, containing the report of the Committee of Medical Inspection of Schools of the American Medical Association, excerpts from the Pennsylvania School Code covering the subject matter of school sanitation, and the Massachusetts Act restricting the use of common drinking cups. School furniture and ventilation are intelligently discussed.

The section on infectious and communicable diseases gives the ordinary methods of transmission of contagious diseases, adminis-

trative considerations in the schools, including the exclusion of children and the quarantine periods. The publishers have furnished most excellent colored plates of vaccination pustules, sore throats of different types (follicular tonsillitis and diphtheria), and Koplik spots in measles.

The section on physical defects includes the consideration of the eyes, ears, nose, and throat, orthopedic defects, systemic diseases, and mentality. These subjects are all well treated, much better than in the average book. The chapter on the teeth is particularly good. The illustration showing objects as they appear to a child suffering from astigmatism produced by the insertion of a cylindrical lens before the camera is hardly true to nature, as it takes no account of that improvement in the sight of such individuals which results from the repeated changing of the focus and from differences which frequently exist in the axis of astigmatism of the two eyes.

The subject of mentality is very fairly written, the usual causes of mental retardation being stated, and also the Binet-Simon tests given are fully reported. This chapter, however, is not quite up to the general standard of the book, the term "feeble-minded" being used exclusive of imbeciles and idiots, and no distinction being made between the feeble-minded who are degenerate and those who are in such condition from accidental brain injuries. In justice to this chapter it should be said that it does present the matter to a non-expert reader in such a way that familiarity and confidence are obtained.

The book closes with the citation of questions asked in civil service and other competitive examinations for the position of school medical inspector, valuable to physicians considering entrance into public health work. W. S. C.

CANCER: ITS STUDY AND PREVENTION. By HOWARD CANNING TAYLOR, M.D., Gynecologist to the Roosevelt Hospital, N. Y.; Professor of Clinical Gynecology, Columbia University. Pp. vi, 331, Philadelphia: Lea & Febiger.

THE author aims to give the practitioner a summary of the most important facts now at hand concerning malignant neoplasms, to place such knowledge as is of practical value in the handling of these cases in an easily accessible form, and to point out how important it is that greater care be taken by the practitioner in collecting accurate statistics upon the cancer problem in order to further our knowledge of the subject.

The first third of the book is devoted to the etiological factors apparently influencing the development of cancer, to the statistical

evidence of its increasing frequency, to theories of its nature, and to a discussion of its contagiousness and of the nature of metastasis, recurrence, and cachexia. The subject is treated from the clinical aspect with only occasional references to the recent experimental work. This is no doubt because the experimental work has not yet reached a stage which permits of many deductions of practical importance to be drawn from it. On the other hand, barring this experimental work, little that is new has been added to the general problem of the malignant neoplasm in recent years, so that this portion of the work is for the most part a restatement of the views that have prevailed for many years. The latter two-thirds of the book is devoted to a systematic presentation of the etiology, pathology, symptomatology, diagnosis, and treatment of carcinoma and sarcoma of the various organs of the body—being a considerably fuller account of this portion of the subject than is given in the general text-book of medicine, and being thoroughly up-to-date. J. H. A.

THE MEDICAL CLINICS OF CHICAGO. By VARIOUS CONTRIBUTORS. Vol. I, Nos. 1, 2 and 3. Pp. 605. Philadelphia and London: W. B. Saunders Company, 1915.

THE *Medical Clinics of Chicago* have manifestly been brought out for the purpose of occupying in the medical world a position similar to that which is held by Murphy's *Clinics* in the surgical field. They follow the same general plan as do their surgical predecessor and in many ways are entirely similar. They lack, however, one of the strongest appeals of Murphy's *Clinics*, the dominant personal touch of a strong and brilliant man whose personality pervades each page. This is not said with any idea of disparaging the several contributors to the *Medical Clinics*, for it is obvious that among ten contributors some of their presentations would be lacking in something and would fall behind the others in general merit, thus weakening the general value of the whole work.

The plan of the clinics is to present a case, as in any clinic, to demonstrate it, and from this case to draw conclusions and likewise to use it as a text for a general discussion of the particular disease from which the patient is suffering. Ordinary clinics are of undoubted worth; they impress the hearers through their sense of sight and of hearing. Clinics presented in writing lack that appeal to the eye and ear which makes a clinic of value. They have all the faults of an ordinary clinic without the attending circumstances which mitigate the shortcomings of the clinic that is heard and seen. Likewise a carefully prepared paper written with thought and consideration,

without irrelevant interruptions, can be read with far greater value by the physician than a stenographical report of what a man has spoken extemporaneously. If then such articles lack the appealing elements of a seen clinic and the knowledge to be derived from the perusal of a scientific paper, in what does their value consist? The answer to this question is that this work will be bought and read by the man who does not care to read scientific papers, by the man who has not the opportunity of seeing clinics, by the man who likes discussions of every-day problems, and by the man who is so tired after a hard day's work that he is unable to follow the conventional medical publications with any degree of concentration. To such men the medical clinics will have a very real value.

The contributors to the past three numbers of these clinics include Drs. I. A. Abt, M. L. Goodkind, W. W. Hamburger, R. C. Hamill, C. L. Mix, R. B. Preble, W. A. Pusey, F. Tice, R. J. Twosen, and C. S. Williamson.

If it is conceded that such a work has a certain place to fill in medical writings, a better list of contributors would be difficult to secure in Chicago or in fact in any large city. J. H. M., Jr.

A Guide to the Use of Tuberculin. By Archer W. R. Cochrane, M.B. (Lond.), F.R.C.S. (Eng.), Major in Indian Medical Service; Medical Superintendent, King Edward VII Memorial Sanatorium, Bhowali, United Provinces; and Cuthbert A. Sprawson, M.D., B.S. (Lond.), M.R.C.P., Major in Indian Medical Service, Professor of Medicine, King George's Medical College, Lucknow; Physician to King George's Hospital and to the Lucknow Tuberculosis Hospital. Pp. 181; 84 charts. New York: William Wood & Co., 1915.

This book is an attempt on the part of two men who have had a large experience in the use of tuberculin to reduce to writing the general rules which have guided them. They regard fully 75 per cent. of the cases of pulmonary tuberculosis coming to them as suitable for tuberculin treatment, and they do not consider a secondary infection by other organisms or a hemoptysis as a contra-indication. They do, however, regard as contra-indications marked emaciation, tuberculous diarrhea, a very rapid pulse, a high and continued fever, and acute disease. Special emphasis is placed upon temperature and weight records as guides to the continuation of treatment, the proper interval between doses, and the proper doses. The charts displayed in the book are numerous and most instructive. Only enough theory is given to make appreciable the clinical and pathological aspects of tuberculin therapy. While

the number of cases which they regard as suitable for tuberculin injections seems large, there is evident a marked conservatism in their method of applying the treatment, and the thought is forced upon us that some of our failures may have been due to faulty application. T. G. M.

THE MENTAL HEALTH OF THE SCHOOL CHILD. By J. E. WALLACE WALLIN, PH.D., Director-Elect of the Psycho-Educational Clinic, St. Louis Public Schools. New Haven: Yale University Press.

IN this book the author has endeavored to cover the field of clinical psychology in its modern aspects, correlating it with school medical inspection and the physical health of the child, and to furnish a compendium of information concerning organized activities in this field throughout the United States.

The book contains nineteen chapters which in brief include the consideration of clinical psychology, the functions of the psychological clinic, psychological research among epileptics (in which group Dr. Wallin has had much valuable special experience), the present status of the Binet-Simon tests, and the relation of physical health to mental development, including special consideration of the relation of carious teeth to mental development.

There is also presented a skeleton outline for the recording of case histories when dealing with supposedly deficient children, the field of investigation covering social, medical, psychological, and educational information.

Taken together, the book is full of interesting facts, gathered evidently at great labor and correlated by one with a keen analytical mind. The book is really one of the most valuable contributions of solid material to those interested in mentally deficient children. Extensive bibliographies are given at the end of several of the chapters, and the appendix contains a mass of statistical information concerning the work that is being done by educational authorities in different American cities, the places at which instruction is given in the subjects, and the cities in which special instruction for tuberculous, blind, crippled, etc., children is given.

The most valuable features of the book are the commentaries upon the value and limitations of the Binet-Simon psychological tests, the mental condition of epileptics, and the great mass of information, statistical and otherwise, concerning the education of deficient children throughout the United States.

The only points in the book which may be reviewed in a critical spirit are the chapter on the relation of decayed teeth to the mental development, the repetition of the writer's views upon the limits of the Binet tests, and the tendency to use language which, while

thoroughly sound and scientific, might be replaced by simpler English words.

Regarding the relation between dental caries and mental development the conclusions are possibly premature. In the matter of the limits of the Binet tests the reviewer agrees with the author absolutely in his conclusions, but does not feel that the claims of the ardent advocates of these tests as a diagnostic measure are worth combating so constantly.

Altogether, the book is one that every person giving special study to the subject of mentally deficient children would do well to possess. W. S. C.

MANUAL OF SURGERY. By ALEXIS THOMSON, F.R.C.S. (Edin.), Professor of Surgery, University of Edinburgh; Surgeon to Edinburgh Royal Infirmary; and ALEXANDER MILES, F.R.C.S. (Edin.), Surgeon to Edinburgh Royal Infirmary. Fifth edition; two volumes. Pp. 1724; 590 illustrations. London: Henry Frowde; Hodder & Stoughton, 1915.

THESE two volumes comprise two-thirds of the work by these authors on surgery. One volume considers general surgical questions, the second takes up regional surgery, and the third treats the operative side of all surgery. In many ways this arrangement is an advantage, making it much handier and convenient in reference.

This, the fifth edition, has improved the entire work. The subject matter on pathology and treatment has been brought up to date. Many new illustrations have been added and all debatable questions left out entirely. The arrangement of the subject matter is very good. The paragraphs on surgical anatomy that frequently head the chapters are a great help and save much time when one is making a detailed study of the subject. The idea is an excellent one and is especially serviceable to students.

The text is remarkably free from useless references and bibliography. No space is given to theorizing. Facts are stated when definitely known. Each subject is treated in its entirety, *i. e.*, its etiology, symptomatology, complications, diagnosis, and treatment.

The style of the text is very pleasing, the expression to the point, clear, concise and smooth. The illustrations are good and really illustrate their subject. In some cases, however, the stage of the disease shown seems a little far advanced. Diagnosis should be made early and illustrations should aid in doing this rather than in showing final stages of disease.

The book itself is well put up. It is handy in size, the print is clean and easily read, and the paper all that could be desired.

The work is a good one. E. L. E.

DIFFERENTIAL DIAGNOSIS. By RICHARD C. CABOT, M.D., Assistant Professor of Clinical Medicine, Harvard University Medical School, Boston, Mass.; Chief of the West Medical Service, Massachusetts General Hospital. Vol. II. Pp. 709; 254 illustrations; 22 charts; one colored plate. Philadelphia and London: W. B. Saunders Company.

THIS new volume of Dr. Cabot's work is modelled along the lines of the well-known earlier volume. The author presents the clinical history, physical examination, and laboratory findings of a series of illustrative cases, discusses the diagnosis on the basis of these data, and then gives either the autopsy findings or the diagnostic evidence from the further course of the case. No more vivid means of presenting bedside diagnosis in book form can be conceived, and the careful study of the material which he presents is exceedingly instructive as well as entertaining. Each of the nineteen chapters is devoted to cases exhibiting some particular symptom or physical finding as follows: abdominal and other tumors, vertigo, diarrhea, dyspepsia, hematemesis, glands, blood in the stools, swelling of the face, hemoptysis, edema of the legs, frequent micturition and polyuria, fainting, hoarseness, pallor, swelling of the arm, delirium, palpitation and arrhythmia, tremor, ascites, and abdominal enlargement. In all 317 cases are detailed. At the beginning of each chapter the author devotes a few pages to a discussion of the general subject of the chapter, and presents in chart form some statistical material concerning the relative frequency with which various conditions give rise to the disorder under consideration. The work is freely illustrated with temperature charts, diagrams of the physical findings, blood charts, etc. J. H. A.

THE GOLD-HEADED CANE. By WILLIAM MACMICHAEL, M.D., with an introduction by WILLIAM OSLER, B.A., M.D., F.R.S., and a preface by FRANCIS R. PACKARD, M.D. Pp. 254. New York: Paul B. Hoeber, 1915.

THE present edition of the *Gold-headed Cane* is gotten up in an attractive and tasteful manner. The brief biographical sketches of Radcliffe, Mead, Askew, Pitcairn, and Baillie, as presented in this volume, will doubtless be welcomed by all interested in historical medicine. J. H. M., JR.

PROGRESS

OF

MEDICAL SCIENCE

SURGERY

UNDER THE CHARGE OF

J. WILLIAM WHITE, M.D.,

FORMERLY JOHN RHEA BARTON PROFESSOR OF SURGERY IN THE UNIVERSITY OF PENNSYLVANIA AND SURGEON TO THE UNIVERSITY HOSPITAL,

AND

T. TURNER THOMAS, M.D.,

ASSOCIATE PROFESSOR OF APPLIED ANATOMY AND ASSOCIATE IN SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE PHILADELPHIA GENERAL HOSPITAL AND ASSISTANT SURGEON TO THE UNIVERSITY HOSPITAL.

Partial Occlusion in the Treatment of Aneurysms.—MARTIN (*Surg., Gynec.* and *Obst.*, 1915, xxi, 629) reports 3 interesting cases of pulsating exophthalmos, due to trauma, and completely cured by ligation of the common carotid artery alone in 1 case and partial ligation of this artery and some orbital veins in 2 cases. The treatment indicated in these cases, due to arteriovenous aneurysm, would be, after preliminary tests as to the effect of compression of the carotid: (1) An occlusion of the vessel by an absorbable ligature up to the point of stopping the bruit and pulsation. The patient can best testify as to the disappearance of the bruit but must distinguish between a true ear tinnitus which often follows basal fracture and the rythmically throbbing noise of his aneurysm; (2) ligation of one or more dilated orbital vessels; (3) starvation diet, guarding against acidosis, with but little fluid; bowel evacuations, accomplished without straining, and two weeks' rest in bed made, if not alluring, at least bearable by the judicious use of anodynes.

Typhoid Perforation.—GIBBON (*Ann. Surg.*, 1915, lxii, 385 and 539) collected and studied 139 cases from the records of the Pennsylvania Hospital. He believes that our errors result not from any lack of knowledge of symptoms, but rather from a hesitancy to comprehend the importance of promptly acting when these symptoms present themselves. The two most valuable symptoms are pain and rigidity, and on these symptoms we must rely nearly altogether. Of course, if the pain comes on suddenly and the rigidity is marked, a perforation must be suspected and the exploration made. The less sudden and severe the pain and

rigidity, the more difficult the diagnosis becomes. Digital examination per rectum should always be made and often will reveal the very significant acute tenderness. The absence of a leukocytosis is of no diagnostic value. As a rule it takes from eight to twelve hours for the average case of perforation to develop a leukocytosis. The presence of a leukocytosis, of course, is of diagnostic value, There should be no delay in operating if the surgeon believes from the symptoms that a perforation has probably occurred. A quick exploration does not do much damage, but delayed operation is the cause of the high mortality. The safest rule regarding anesthesia, is to open the abdomen under infiltration anesthesia and administer a general anesthetic only if it is necessary, and then for as brief a time as possible. The rule in Gibbon's cases was to close the perforation with drainage of the abdomen but in a number of cases the perforated bowel was sutured in the abdominal wound and allowed to drain. Continuous enteroclysis is the most important part of the after-treatment. The Fowler position, although of great help in many cases of perforative peritonitis, is not always advisable in typhoid fever, as the patient is often too weak to stand it.

Fractures of the Femur.—STEINKE (*Ann. Surg.*, 1915, lxii, 610) says that much has been written concerning the fractures of all bones but the femur presents some of the greatest difficulties to be encountered while its anatomical and physiological importance accentuates the necessity for a good result. Steinke followed up the late results of 62 cases taken from the records of the Episcopal Hospital, 15 of the fractures occurring through the neck of the femur, 6 through the trochanters, 36 through the shaft and 5 through the lower end. As to treatment, the non-operative gave as good results, functionally, as the operative, although the plated cases were selected, which without operation most certainly would have given poor results. Good approximation and alignment of the fragments is not necessary for a straight and unshortened limb. Fractures of the neck were more frequent in the aged of the female sex, indirectly cause a high mortality because of the age of the patient and the lowered resistance, and gave a moderate disability period. Fractures through the trochanters were all in males and gave a long disability period with lowered earning capacity in two-thirds of the cases. Fractures of the lower end of the femur gave an average disability of about six months, while a good functional result was obtained in about one-half of the cases. They were all males. Fractures of the shaft gave the best results and had the shortest disability period. They were the most frequent and were mostly males. The mortality from fractures of the femur other than of the neck was low, being about 2 per cent. in this series.

Obturator Hernia.—MCMAHON (*Ann. Surg.*, 1915, lxii, 710) says, quoting Meyer, that the diagnosis at best is only a probable one and that operation should therefore be early. He reports a case operated on in which, although the patient said he had a hernia for four years and gave the signs of having suffered from strangulation for a week, the hernia could not be located until the abdomen had been open. It could not be reduced until after exposure of the hernia in the obturator

region. Recovery was uneventful; the bowel after the reduction of the hernia having no evidence of circulatory impairment. In view of the difficulty of positive diagnosis and the difficulty of exposing the obturator region by dissection, a preliminary median abdominal laparotomy seems a sensible procedure in suspected cases of obturator hernia. It has the following points of advantage: The diagnosis is cleared up at once. The possibility of making an exposure of the obturator region only to find no hernia there is avoided. The abdominal incision greatly facilitates any necessary repair of the herniated intestine. In cases where the condition of the patient demands quick work, the hernia can be reduced from within the abdomen, relieving the constriction under direct inspection with the patient in the Trendelenburg position. The obturator opening could then be closed by a few sutures from within. When the patient's condition permits, the obturator region can be exposed and the sac dissected free and ligated. Inasmuch as it establishes the diagnosis at once and facilitates subsequent operative procedure, preliminary laparotomy seems extremely rational. The modern system of prompt laparotomy in the early stages of all cases of intestinal occlusion will, no doubt, very much lower the mortality-rate in future cases of obturator hernia.

The Action of Radium on Transplanted Tumors of Animals.—Wood and Prime (*Ann. Surg.*, 1915, lxii, 751), working under the George Crocker Special Research Fund at Columbia University, used rat and mouse tumors of various types, among them the Ehrlich spindle-cell mouse sarcoma, the Flexner-Jobling rat carcinoma, and mouse carcinomata No. 11 and No. 180 of the Crocker Fund series. These were treated either after removal from the host or *in situ*. In the former method a portion of the tumor to be used was cut into small pieces of uniform size, and these were moistened with Ringer's solution, put into hollow slides, covered with a thin cover-glass, sealed with paraffin, and exposed to the tubes of radium. Their conclusions are as follows: Three factors only are important in the action of radium on tumors: time of exposure, amount of radium element, and distance between the radium tube and the tumor tissue. The removal by suitable filters of the larger part of the beta rays diminishes proportionately the effect of the radium, but the effect of the gamma rays is in accordance with the same general law which governs the beta rays. Sublethal exposures slow the growth of tumor cells for some time, while shorter treatments seem to stimulate the cellular activities. The facts derived from their experiments regarding the quantity of radium element and the time of exposure necessary for a given distance may be applied, with reasonable accuracy, to human malignant tumors. The experiments show also that when only pure gamma rays are used the necessary exposure is eight times as long as that required when the gamma and hard beta rays combined are employed, but as the latter are largely absorbed by 1 cm. of tissue the gamma rays alone must be used for all the deep work. The effect of the radium radiations on tumor cells *in vitro* is less marked than is that on isolated cellular elements. This explains the fact that an exposure which will destroy a small metastatic nodule in man is quite ineffective in the case of a well-vascularized primary carcinoma.

THERAPEUTICS

UNDER THE CHARGE OF

SAMUEL W. LAMBERT, M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE COLLEGE OF PHYSICIANS AND SURGEONS, COLUMBIA UNIVERSITY, NEW YORK.

The Effect of Carbon Dioxid in the Inspired Air on Patients with Cardiac Disease.—PEABODY (*Arch. Int. Med.*, 1915, xvi, 846) describes experiments in which normal individuals and patients with cardiac or cardiorenal disease have been made to breathe air containing increasing amounts of carbon dioxid. The carbon dioxid content of the inspired air was determined for each subject, and the total ventilation of the subject per minute was measured at frequent intervals. Peabody found that normal individuals react to carbon dioxid in a fairly constant manner, and that their total ventilation is doubled when the carbon dioxid content is between 4.2 and 5.4 per cent. of the inspired air. Patients with cardiac and cardiorenal disease well compensated, who are without acidosis as indicated by the carbon dioxid tension of the alveolar air, react in a manner similar to normal subjects. However, patients suffering from these same diseases, but with decompensation and with acidosis are much more susceptible to the carbon dioxid in the inspired air. Dyspnea is more easily produced than in normal subjects or in patients without acidosis and the ventilation may be doubled when the inspired air contains only 2 to 3 per cent. of carbon dioxid. That the increased sensitiveness to carbon dioxid does depend on the acidosis was clearly demonstrated in 2 cases. Peabody says that while acidosis is not the only factor causing the dyspnea in cardiac and cardiorenal disease, it may be an element of considerable importance in producing the clinical picture of decompensation. The article suggests strongly as a therapeutic measure to relieve dyspnea, the correction so far as possible of acidosis occurring in patients suffering from cardiac or cardiorenal disease.

The Acidosis of Chronic Nephritis.—PEABODY (*Arch. Int. Med.*, 1915, xvi, 955) says that in mild cases of uncomplicated chronic nephritis, in which the phenolsulphonephthalein test shows a normal renal function there is usually little or no acidosis. More advanced cases, showing moderate or even extreme decrease in the phenolsulphonephthalein output, show an acidosis by the "alkali tolerance" test, but there may be no fall in the alveolar carbon dioxid tension. Only in very advanced cases is the acidosis usually so marked as to cause a decrease in the alveolar carbon dioxid tension. Most cases in which the alveolar carbon dioxid tension is below normal show a phenolsulphonephthalein output which is below 10 per cent. in two hours. On the other hand, cases showing a phenolsulphonephthalein output of "traces" or less may have a normal alveolar carbon dioxid tension. The acidosis of chronic nephritis is due to a retention, resulting from inefficient renal

excretion. Acidosis is probably a very constant feature of uremia, but only in a limited number of cases is it of sufficient grade to cause definite clinical symptoms analogous to those seen in advanced diabetes. In these cases the symptoms caused by the acidosis may be relieved by alkali therapy.

The Treatment of Amebic Dysentery.—Low (*Brit. Med. Jour.*, 1915, 2863, 714) says that emetin may be regarded as a specific drug in the treatment of the amebic form of dysentery and can be given intravenously, subcutaneously, intramuscularly, or orally. Intravenous administration may be adopted in moribund or fulminant cases, but the other methods are preferable for general use. The subcutaneous injections often result in local pains and stiffness which may last for several days, and the same effects may follow intramuscular injections, but are less frequent. Intramuscular injection is the most suitable method of administering emetin, but care should be taken to avoid giving successive doses into the same region. Intramuscular and subcutaneous injections are not followed by nausea or vomiting, even when large doses are given, but these symptoms may result from oral administration. So far as curative effects are concerned, there is no choice of mode of administration so long as the drug is retained. For oral administration the drug should be given in keratin-coated tablets at bedtime. For injection the dose should be two daily injections of a grain each for the first few days, and thereafter one grain every evening until a total of 12 grains have been given. If the symptoms are not all relieved, the disease is probably due to some cause other than the ameba. The action of the drug on the vegetative or living stage of the ameba is very rapid, the parasites quickly disappearing, as do also the blood and mucus and other symptoms. Emetin is said not to effect the ameba in its encysted form, but in the experience of the author daily doses of 1 grain each quickly caused these forms to disappear from the stools. The treatment of the chronic cases and carriers is therefore the same as for the acute cases. Bed treatment is not now necessary, but careful attention to the diet and a systematic course of emetin is essential to a cure. Low notes that the incidence of liver abscess complicating amebic dysentery is much less with emetin treatment.

Artificial Pneumothorax in the Treatment of Pulmonary Tuberculosis.—Sachs (*Jour. Amer. Med. Assn.*, 1915, lxv, 1861) reviews critically the results obtained by twenty-four American observers in the treatment of pulmonary tuberculosis with artificial pneumothorax. The author in his conclusions states that experience with artificial pneumothorax in the treatment of pulmonary tuberculosis is gradually modifying the sphere of its application. The trend is toward its use, regardless of stage, in all progressive cases (particularly with unilateral involvement) which fail, after sufficient trial, to respond to strict sanatorium regimen. Complications incident to the employment of this method, the great importance of continuous strict observation and thorough clinical, laboratory and Roentgen study of each individual case, the period essential after each reinflation, the inability of outlining in advance the time of reinflations, in short, the close dependence of each

successive step in pneumothorax therapy on the varying condition of the patient, are all factors which call, in the majority of cases, in the interest of safety and best results, for hospital or sanatorium supervision, at least for a few months, of each individual case in which the method is being applied. Close attention to every important detail of technic, as well as strict asepsis, is also less of a problem, under such conditions, regardless of the excellent results obtained by some observers in dispensary and office practice. The present tendency, grown out of experience, is toward greater conservation in the selection of cases, a selection based on thorough preliminary study and full consideration of the possible effect of compression in the individual case. Conservatism needs to characterize each successive step in the gradual attainment and subsequent maintenance of a satisfactory compression, with avoidance of too frequent reinflations and excessive intrapleural pressure. This attitude is very important, with a method the application of which cannot be subject to rule, but is dependent on the condition of each individual case. Through improvement of various details of technic and greater conservatism of action, pneumothorax therapy is gradually finding its defined place in the treatment of cases of pulmonary tuberculosis which do not yield to other methods.

Observations on Antityphoid Vaccination.—NICHOLS (*Jour. Exper. Med.*, 1915, xxii, 780) says that the living sensitized typhoid vaccine is not without danger for general use, especially in the military service, as it retains the power of causing typhoid fever if accidentally taken by mouth. He also points out the non-relationship between virulency and toxicity. The army strain, although pathogenic and relatively avirulent nevertheless is distinctly toxic, and it is upon this latter factor that its efficacy depends. In regard to Gay's typhoidin skin reaction, Nichols believes that it is not an index of true immunity, but rather an indication of typhoid protein sensitization, which is not as complete, permanent or specific as true immunity.

The Late Results of Active Immunization with Diphtheria Toxin-Antitoxin and with Toxin-Antitoxin Combined with Diphtheria Bacilli.—PARK and ZINGHER (*Jour. Amer. Med. Assn.*, 1915, lxv, 2216) in an earlier report found that the cases injected could be divided into two sharply differentiated groups: (1) Those who had a natural antitoxin immunity produced a very decided increase in the amount of antitoxin after injections of toxin-antitoxin. The response was very prompt and followed even after a single injection. These individuals were, however, naturally immune even before the injections, and needed no further immunizing injections. (2) Those who had no natural antitoxin immunity and were therefore probably susceptible to diphtheria gave entirely different results. An early response (within from four to six weeks after the injections) to the active immunization was noted in only 25 to 30 per cent. of the injected individuals. The examination of the blood serum of these patients for antitoxin showed the presence of from one-tenth unit to ten units per cubic centimeter. Among those who did not definitely respond, however, a number gave a fainter Schick reaction than the control test made before the injections of toxin-antitoxin. This showed that at least a

trace of antitoxin had developed in the cells and had accumulated in the blood. When these individuals were retested from six to eighteen months later, a large proportion now gave a negative Schick reaction and were thus found to have finally become immune. Their results in active immunization up to the present time have led the authors to the conclusion that, while the toxin-antitoxin injections cannot be depended upon to prevent diphtheria in the presence of immediate danger of infection because of the slow development of immunity in a majority of cases, yet the injection is of value in rendering a community or school population immune to the dangers of infection from future exposure up to a period of possibly several years. The authors conclude from their experience with these injections that individuals who, before treatment, give a negative Schick reaction are immune probably for life and, therefore, it is not necessary to inject them, when exposed, either with antitoxin or toxin-antitoxin. Those who give a positive Schick reaction and are exposed to diphtheria and in immediate danger should receive either antitoxin alone or, if a longer protection is desired, both antitoxin and toxin-antitoxin. For the general prophylaxis against diphtheria in schools and communities, excluding immediate contacts, a mixture of toxin-antitoxin alone (from 85 to 90 per cent. of the L+ dose of toxin to each unit of antitoxin) or toxin-antitoxin plus vaccine of killed diphtheria bacilli is recommended. The dose is 1 c.c. of toxin-antitoxin and 1,000,000,000 bacteria injected subcutaneously and repeated three times at intervals of six or seven days. Sufficient time has not as yet elapsed to judge the value of adding the injections of the bacilli to the toxin-antitoxin. The early and the late results of active immunization should be determined with the Schick test. Early results are those obtained by the application of the test within four weeks, and late results from four months to two years after the immunizing injections.

The Treatment of Paresis by Injections of Neosalvarsan into the Lateral Ventricle.—HAMMOND and SHARPE (*Jour. Amer. Med. Assn.*, 1915, lxv, 2147) present a preliminary report dealing with the injection of salvarsanized serum and small doses of neosalvarsan directly into the lateral ventricle for the treatment of general paresis. The report shows that the operation can be successfully performed and they believe that further experience with selected early cases will show a decided superiority to other methods of treatment from a clinical standpoint. The authors state that, in comparison with the deadly nature of paresis, the hazard of intradural treatment, by whatever method, is of little moment. On experimental and clinical grounds both subdural and intraventricular methods are superior to the intraspinal route, in the treatment of paresis. They believe, furthermore, that the intraventricular method is not only superior to the subdural route, but also safer. The intraventricular method, with careful technic, and a due regard for the anatomy of the brain and the delicate nature of the tissues is practically free from danger. The authors are of the opinion that if the freedom from unfavorable symptoms so far achieved in intraventricular injection can be maintained, it will be imperative to so treat paresis in its earliest stages, with greater chance of marked improvement and perhaps permanent arrest of symptoms.

The Treatment of Syphilis of the Nervous System by Intradural Injections of Salvarsan.—Wile (*Jour. Lab. and Clin. Med.*, 1915, i, 119) reports fifteen cases of various forms of syphilitic disease of the central nervous system treated by intradural injections of old salvarsan. None have suffered any ill consequences. In all but 2 patients definite objective improvement could be noted in the spinal fluid. With full allowance for the possible suggestive element, there was, nevertheless, a marked subjective improvement in the majority of the cases. Such improvement was not seen to be transitory, but appears to be permanent. Three patients have disappeared from observation, but none of the remaining 12 have relapsed with regard to symptoms. Four patients have been restored to usefulness and are making a livelihood, which before their treatment they were unable to do. The patients showing the most marked improvement were those suffering from early brain or cord syphilis, but encouraging results were also noted in cases of tabes dorsalis. No patient received over three injections, but it seemed to the author that further treatment in cases in which improvement had occurred would be indicated.

The Reflex Effects of Alcohol on the Circulation.—Lieb (*Jour. Amer. Med. Assn.*, 1915, lxiv, 898), upon the ground of experimental and clinical observations, says that small doses of undiluted whisky cause a reflex effect on the circulation in a certain percentage of the patients. The reflex is most evident in those cases with hyperactive superficial and deep reflexes, least evident in those who are apathetic as a result of bacterial toxemia. It is more marked in moderate users of alcohol than in those who use alcohol to excess. This is probably due to the protective secretion of mucus which is present in alcoholic gastritis. The reflex is elicited only by irritating concentrations of whisky. Well-diluted whisky causes practically no changes in the circulation before absorption. The degree of response varies more or less directly with the degree of irritation induced; that is, with the dose and the concentration. The changes produced in the heart rate are so small that they are well within the normal fluctuations. The systotic blood-pressure is raised only slightly. The maximum is reached within the first minute, and there the pressure declines so that it reaches normal in less than half an hour. The diastotic pressure is also raised, proportionately less than the systolic, but the return to the original level is very much slower. As a result, the pulse pressure, which was temporarily increased immediately after the whisky was swallowed, is permanently decreased. That is, the pulse after a fleeting increase in size becomes smaller. In some individual cases the decrease in pulse pressure is brought about by a disproportionate rise in the diastotic pressure as well as by its comparatively slower decline. From these investigations it is apparent that alcohol does not stimulate the circulation either by increasing the heart rate or by raising the blood-pressures. The advocates of the use of alcohol, while admitting the truth of the foregoing, may claim that whisky improves the circulation in some other fashion. Thus, this drug may increase the work done by the heart or it may increase the velocity of the blood flow or it may improve the efficiency of the heart as a pump. Lieb found that the changes induced by whisky in these three factors were comparatively insignificant.

Acute Yellow Atrophy of the Liver in Syphilis.—Bendig (*München. med. Wchnschr.*, 1915, lxii, 1144) reports a case of primary syphilis who was being treated with both mercury and salvarsan. The first two injections of salvarsan were followed by no untoward symptoms, but the third injection produced marked vomiting, and two days later symptoms of acute yellow atrophy developed with a fatal termination within two days. Bendig calls attention to the fact that he reported in 1908 an almost identical fatal case. Since this was before the discovery of salvarsan he believes that salvarsan need not necessarily be the cause of death in the present case.

PEDIATRICS

UNDER THE CHARGE OF

THOMPSON S. WESTCOTT, M.D., AND FREDERICK O. WAAGÉ, M.D., OF PHILADELPHIA.

Tuberculous Skin Lesions.—Northrup (*Arch. Pediat.*, 1915, xxxii, 887) reports a tuberculous skin eruption in a child of fifteen months. Three similar cases were reported previously. This condition is rather rare in the very young and Tileston concludes it occurs only in fatal cases as a terminal event. The case in point had tuberculous meningitis and pulmonary tuberculosis besides the skin lesions. There was a history of tuberculosis in the mother and a brother. The history shows progressive wasting for six months. The lymphatic glands of the neck, axilla and inguinal region were moderately enlarged. The skin eruption was mostly on the trunk, especially on the back, some on the face and forearm. The character of the lesions was papulopustular and umbilicated, and while showing no tubercle bacilli on microscopic examination, was diagnosed as a tuberculide of the skin with some secondary infection. There were tubercles in the lymph nodes and subcutaneous tissue, the lungs, meninges, and intestines. While no bacilli were found in the sputum, glands or skin lesions, the Roentgen-ray showed miliary tuberculosis of the lungs. The physical signs in the chest had been inconclusive. This exemplifies the important bearing the Roentgen-ray has on diseased conditions, especially in children. While localized tuberculosis is common, disseminated tuberculous eruption of the skin, necrotic tuberculid, is rare in infancy.

Macewen's Sign in Intracranial Pressure.—Wilcox (*Arch. Pediat.*, 1915, xxxii, 909) offers an interesting elucidation of Macewen's sign in changes and intracranial pressure and illustrates the practical application of this change of note. Different normal percussion notes belong to varying ages, with their corresponding states of skull development. Disease influences the character of skull percussion, rickets being the best example of this. A rachitic skull at six years

will give a percussion note similar to that found normally in an infant of six months. If this case develops increased spinal fluid a sharp, high-pitched, clear note is at once found to have replaced the former full, low-pitched, non-resonant percussion. If the cranial walls are too thin no note will be produced even though intracranal pressure be high. Macewen's sign is best determined by the stethoscope placed on the forehead just above the base of the nose. The skull is tapped directly with the percussing finger or hammer over the parietal region, beginning just over the parietal boss, from which the finger approaches the stethoscope. This should be carried out on both sides of the head. The typical sign observed in this way consists of a high-pitched, sharp, short, cracked-pot note. It is most distinct over and around the parietal boss and diminishes toward the stethoscope, while the reverse obtains in a normal skull where the note is heard more loudly as one approaches the stethoscope. The cracked-pot note of Macewen should be considered as a relative variation from the normal and not as a definite entity. When this sign is positive and cerebral fluid under pressure is withdrawn in the usual amount the note goes back to more nearly the normal almost at once. The sign was present in 50 of 53 cases of tuberculous meningitis and 17 of 18 cases of meningitis of other types. It was present in all of 5 cases of poliomyelitis. It was found to vary directly with the development and recession of cerebral symptoms as complications of disease not directly affecting the central nervous system. The sign is uniformly lacking in children normal as to the brain and its coverings.

Infantile Paralysis in North Austria.—STIEFLER (*Wien. klin. Wchnschr.*, 1915, xxviii, 1079) states that aside from very occasional sporadic cases, anterior poliomyelitis did not appear in epidemic form in Austria, especially the northern part, until 1907. Stiefler analyzed 187 cases occurring from 1909 to 1913. These cases are practically equally distributed among males and females. The highest number of cases occurred in children from three to six years of age (45 and 46 cases), the next highest being between four and six years (32 cases). The incidence decreases with increasing age periods, but between forty-one and fifty years 9 cases are reported. Most of the cases occurred among the farming and laboring classes. Almost all cases showed a prodromal stage of several days with fever, rarely chill, as the leading symptom followed by severe sweats, vomiting, and constipation, and later inflammation of the throat. Otherwise the respiratory system was rarely affected. Extreme tenderness of muscles and nerve trunks and paresthesias with pain in the legs and back are constant symptoms in these cases. Severe delirium occurred in only 11 cases. There was generally a leukopenia. Motor disturbances such as clonic spasm, tremors, and choreiform movements were frequently observed in paralyzed limbs. Under Zappert's classification these cases would be divided as follows: Spinal form, 148 cases; cerebral form, 12 cases; and abortive form, 27 cases. In the acute stage the paralysis affected mostly the lower extremities and in one-third of the cases the gluteal and back muscles were involved. Of 180 cases including the paralyzed and abortive cases 64 recovered completely, 93 recovered partly, and 23 died. Only 37 of the paralyzed cases recovered com-

pletely. The highest mortality occurred between the ages of seven and fourteen years. From the epidemiological standpoint the history of these cases offers no new features. The incubation period was estimated to be from six to ten days.

OBSTETRICS

UNDER THE CHARGE OF

EDWARD P. DAVIS, A.M., M.D.,

PROFESSOR OF OBSTETRICS IN THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

Pregnancy in a Rudimentary Uterine Cornu and in Bicornate Uterus.—MICCABRUM (*Ann. di Osteter*, 1915, No. 8) reports a case from Mangiagalli's Clinic in Milan and illustrates his paper with some excellent colored reproductions of microscopic sections. He finds a special and unusual arrangement of the muscle fiber in the rudimentary cornu in this case and that there is not present the usual arrangements of Langhans's layer. He found a direct connection between the rudimentary cornu and the maternal vessels through the intervillous spaces, nor did the vessels of the rudimentary uterus suddenly terminate in those of the decidua. There is a distinct decidua which forms in these cases and which seems to be developed largely from the muscular tissue. He does not consider that the conditions found are the result of mechanical factors nor that they are secondary to the anatomical peculiarities of the case. His study of the subject convinced him that the data ordinarily given as significant in the diagnosis of these cases are frequently of very little value.

The Origin of Icterus in the Newborn.—BANG (*Arch. mens. d'obstet.*, August, 1915) has studied this subject in the clinic at Copenhagen. He finds that the examination of the blood taken from the skin of the newborn soon after birth shows a great increase in the number of red cells. This must be considered a condition of stasis in the capillaries. During the first few days following birth the percentage of hemoglobin gradually, under normal conditions, returns to the average. Where, however, pathological conditions are present, the excess continues. The conditions are favorable for the retention of the excess in the liver, biliary pigments are formed in excess and absorbed throughout the entire organism.

The Abderhalden Method of Diagnosis in Pregnancy in the Clinic at Basel.—BAUMANN (*Monatschr. f. Geburtsh. u. Gynäk.*, 1915, xlii, 3) gives his experience in the obstetrical wards in the clinic at Basel. He obtained good results with the use of ninhydrin with placental tissue prepared by his own methods and by dialysis. He also had good results with what is known as dried placental albumin manu-

factured by the Hoechst method. Of this he employed 0.5 and 0.25 gm. in his experiment. When he used the moist placental substance, also devised by Hoechst he had 40 per cent. of failures. The substance known as color placental extract failed to give good results. He had especial success by the Abderhalden method in diagnosticating difficult cases where there was much uncertainty in differential diagnosis. Including cases of abortion and tubal molar pregnancy, the percentage of failures in diagnosis was from 3 to 4. If these were excluded, the percentage of failures diminished to 1.5 to 2 per cent. In one instance of tubal gestation, he took the blood direct from the open abdomen and believes that the negative reaction arose from a failure in the technic. In a second case of pregnancy and myoma which gave late negative reaction, it was impossible to tell the reason for the failure of the test.

The Abderhalden Reaction in Patients Who are not Pregnant.—VAN WAASBERGEN (*Monatschr. f. Geburtsh. u. Gynäk.*, 1915, xlii, 3) has made experiments with the Abderhalden test in patients not pregnant. In van der Hoven's Clinic in Leyden the writer examined the sera of 69 women not in the pregnant condition and who had some abnormality or some condition of ill-health. The sera were treated after the Abderhalden method, using the dialyzing apparatus; in order to make a test of the accuracy of the method he employed a 1 per cent. peptone to obtain the characteristic violet color with ninhydrin, before applying the method to the sera in question. The first test made showed that of the 69 sera but 7 gave a negative reaction, but the intensity of the positive reaction differed very greatly. It was interesting to observe that, in cases where there was a weak positive reaction, if the temperature arose, the reaction was very much stronger and that in the 7 cases giving a negative reaction, 3 changed to a positive reaction when fever developed. The 4 sera which remained negative were from patients who had passed the menopause and were suffering from prolapse, while the other 3 sera were from women aged respectively, fifteen, sixteen, and forty-one years. He divided the 68 patients into two groups, one aged from fifteen to twenty-five years; the other twenty-six years and more. In the first group there were twenty and of these 55 per cent. gave a positive reaction; 35 per cent. a weak positive reaction; 10 per cent. a negative reaction. In the second group there were 43 of whom 37.5 per cent. gave a positive reaction; 52 per cent. a weak positive reaction; 10 per cent. a negative reaction. These cases were again studied when some fever had developed and in the first group 90 per cent. gave positive reaction; 10 per cent. a weak positive reaction and none of them a negative. In the older group 50 per cent. gave a positive reaction; 41.5 per cent. a weak positive reaction; 8.5 per cent. a negative reaction. Evidently age had a considerable bearing upon the working of the test. The writer draws attention to LANGE'S (*Berl. klin. Wchnschr.*, 1914, No. 17) experience, who obtained a positive reaction in patients suffering from tuberculosis and in surgical cases where pus had formed. As fever is accompanied by increased metabolism it is natural to expect a positive Abderhalden test when fever is present. The writer in 41 cases obtained the date at which the menstrual period was expected and these patients were

divided into three groups, in accordance with the time elapsing before the actual development of the period. As the time for menstruation approached it was found that the reaction grew more intense, while at the moment of menstruation or immediately after it again became much weaker. Hence the test is a positive indication that pregnancy is not present, but menstruation is approaching.

Disturbance in the Function of the Liver during Pregnancy.—BAUCH (*Monatschr. f. Geburtsh. u. Gynäk.*, 1915, xlii) has examined 22 pregnant patients by the administration of 40 gm. of galactose to determine the presence of this substance in the urine. In 8 (or 37 per cent.) there was the elimination of from 0.4 to 1.8 gm. which did not, however, exceed the amount which is found in healthy non-pregnant cases and should therefore not be taken as indicating a pathological condition. Unquestionably the amount of sugar in the blood increases during pregnancy, but hyperglycemia was not observed. In one case showing some symptoms of a mild pregnancy toxemia there was an increase in the sugar content of the blood and when galactose was administered this became noticeably greater.

Bed-sores Complicating Parturition and the Puerperal State.—KOTTMAIER (*Zentralbl. f. Gynäk.*, 1915, No. 39) reports the case of a primipara, aged twenty-six years, delivered spontaneously who, on the second day after labor, showed in the region of the posterior superior spine of the ileum two bluish-red surfaces which developed into bed sores. The patient had also sustained a laceration of the perineum and stitches had been taken, but this lesion healed slowly and poorly and it was necessary to keep the patient quiet in bed for twelve days. During this time, the bed sore healed under aseptic precautions and the use of ointments. His second case was that of a small, pale, primipara with contracted pelvis, who gave birth to a child in spontaneous labor and in the latter portion of the labor became very excited with greatly increased patellar reflexes and great irritation of the skin. On the second day after the birth of the child in the same region similar lesions developed to those described in the first case. This patient had a gonorrheal endometritis and was three weeks in bed and all of the lesions during that time disappeared. His third case was also a primipara and in vigorous health, delivered spontaneously with considerable laceration of the pelvic floor and perineum. The child and the pelvis were disproportionate and the mother's pains became excessively strong during the latter part of labor. A vesicovaginal fistula developed on the second day which closed on the fifth day with the use of a permanent catheter. After the second day following the birth of the child a reddened area developed over the sacrum which became a bed sore in three days. Its border became well marked and on the sixth day a superficial slough was discharged after which the lesion gradually healed. On the same day when this appeared the patient had left-sided herpes labialis. There was no sudden rise in temperature and the general condition remained good. The reflexes were much increased and there was considerable irritation in the skin. The fourth case was a vigorous primipara with a rachitic and contracted pelvis, not sufficiently small to prevent spontaneous labor. On the day following the birth of the child, there was the development

of a well-defined reddened area over the sacrum and in the two following days, a slough formed and the surface ulcerated which required three weeks' treatment to heal. In these cases, the question naturally arises whether the constant pressure of the abdominal binder can have anything to do with the lesions described. This connection cannot be considered as proven. Those who have reported similar cases have considered them as toxic and that the patient had some predisposition to irritation of the skin which made this occurrence possible. The increased reflexes and irritation in the cutaneous nerves point also to some abnormal state of the nervous system and this is borne out by the fact that these lesions developed in patients who had strong uterine contractions and considerable suffering during labor.

The Indications for Abdominal Cesarean Section.—In a recent book KÜSTNER (*Deutscher Frauenheilkunde*, 1915, ii), in treating of abdominal Cesarean section, naturally considers the indications. First he puts considerable pelvic contraction. This brings the operation into comparison with hebosteotomy, the induction of labor, prophylactic version and the high application of the forceps. As regards repeated section, Küstner does not believe that because a Cesarean section is performed, that sterilization should be done at that time. If a second section becomes necessary, sterilization should also be considered when the uterine scar is found very thin and weak. In his experience, he would often perform the extraperitoneal section for the first and second operations and if the operation was repeated for a third time, he would accompany it with sterilization. He is not in favor, because a pregnant woman has a myoma, of removing the uterus and thus destroying the pregnancy with the removal of the tumor. He believes that Cesarean section is rarely indicated in myoma complicating pregnancy. Rupture of the uterus he believes to be a valid indication for the operation. Should such a patient recover by the suture of the lacerated surface in the uterus, the danger of repeated rupture in a following pregnancy is very great. In cases where a vesicovaginal fistula has required a difficult operation for closure and the patient subsequently becomes pregnant, he would deliver by section when the pelvis is contracted and the fistula was so situated that delivery through the cervix would probably cause the formation of a second fistula. He does not believe that illy developed soft parts or a resisting pelvic floor indicate abdominal Cesarean section. In these cases, as in eclampsia, he would use deep incisions as necessary. In placenta previa complicated by contracted pelvis, profuse hemorrhage and where there is reason to believe that the patient is not infected, Cesarean section would give good results. Many cases of placenta previa are successfully treated by the use of the dilating bag. Abnormalities in the mechanism of labor when they occur in patients having moderately contracted pelves rarely indicate Cesarean section. So, especially if the child is dead, craniotomy should be performed. Where the normally situated placenta becomes misplaced, vaginal section is the operation of choice. As a general rule, abdominal Cesarean section is indicated where it is evidently dangerous and difficult to empty the uterus through the vagina. When the circumstances are thoroughly known and the diagnosis of rapidly impending death is sure, abdominal Cesarean section of the dying may be permitted.

GYNECOLOGY

UNDER THE CHARGE OF

JOHN G. CLARK, M.D.,

PROFESSOR OF GYNECOLOGY IN THE UNIVERSITY OF PENNSYLVANIA, PHILADELPHIA.

Vaginal Supravaginal Hysterectomy for Procidentia.—This operation is advocated by VINEBERG (*Surg., Gynec. and Obst.*, 1915, xxi, 741) for use in those cases where the interposition type of operation would be used were it not for the presence of some contra-indication, such as the presence of fibroids or of uncontrollable uterine hemorrhage, thus necessitating the removal of the uterine body. Instead of doing a vaginal panhysterectomy in these cases, Vineberg does a supravaginal amputation through an anterior vaginal incision, then utilizing the cervical stump as a *pelote* to hold up the bladder. The amputation may be done high enough, if desired, to leave sufficient endometrium for maintenance of subsequent menstruation, the adnexa of course being not removed. If the cervix is greatly elongated, as is frequently the case, Vineberg amputates the lower portion of this as well, leaving only a comparatively short cervical stump, extending from the vaginal vault to the lower uterine segment, to be utilized for the bladder support. After the redundant vaginal flaps are resected, the cervical stump is sutured to the subpubic fascia and the anterior vaginal wall, thus forcing the bladder to take a position within the abdomen, and prevent its prolapse. The operation is completed by repair of the perineum. The author reports having performed this operation ten times, the first over three years ago. Excluding 2 very recent cases, and 1 that was lost sight of, the remainder have been under observation for periods varying from eight months to three years and three months, with excellent anatomic results in all, and equally good clinical results in all but 1. This patient continued to complain of an uncomfortable bearing-down feeling and frequent micturition, although there was no recurrence of the rectocele.

Acute Torsion of the Normal Tube and Ovary.—A case of this extremely rare condition has been recently reported by BARRINGTON (*Jour. Obst. and Gynec. Brit. Emp.*, 1915, xxvii, 141). The patient was a married woman, aged thirty-eight years, who had had twelve children, attended by various complications, the last having been delivered by Cesarean section seven weeks previous to the onset of the ovarian condition. The latter manifested itself by a sudden attack of severe pain in the abdomen while the patient was walking upstairs, gradually getting worse, and localizing to the right iliac region. Almost immediately diarrhea set in, soon followed by vomiting. There was no vaginal bleeding. The pain persisted throughout the night, but was much less severe the following day. On admission to the hospital the patient's abdomen showed no tenderness or rigidity, except on deep pressure in the right iliac region, which did elicit considerable

tenderness. On rectal examination, a lump could be felt above the cervix, independent of the uterus. About twenty hours after the onset of symptoms the abdomen was opened, revealing some excess of clear, not blood-stained fluid in the peritoneal cavity. The appendix was normal, but a tense, elongated lump was left lying behind the uterus to the right of the midline, with its lower extremity at the bottom of Douglas's pouch. On further investigation, this lump was seen to consist of the right ovary, which with the distal part of the tube was twisted on its pedicle at least one complete turn; there were no adhesions to the ovary or uterus. The right adnexa were removed, the left, which appeared entirely normal, being allowed to remain undisturbed. On examination after removal the ovary was found to be about twuce the normal size, its surface uniformly blackish-purple in color; the part of the tube distal to the twist was dark red. A vertical section through the ovary showed the parenchyma to be stained throughout dark red from hemorrhage, but the Graafian follicles and a corpus luteum were preserved and were not invaded by the hemorrhagic infiltration. The question as to any possible etiological relation between the antecedent Cesarian section and the subsequent development of this extremely unusual ovarian lesion is unfortunately not mentioned.

End-results of Nephrectomy for Renal Tuberculosis.—An interesting study of the ultimate fate of patients subjected to nephrectomy for renal tuberculosis has been made by CRABTREE (*Surg., Gynec. and Obst.*, 1915, xxi, 669) from the records of the Massachussetts General Hospital. In a series of slightly over 100 cases there was a primary operative mortality of 4 per cent., with a late mortality of 20 per cent., (about half the deaths furnishing this "late mortality" occurred within two years, and the remainder within five years after operation). The author considers that only about 60 per cent. of the cases can be considered cured of active tuberculous lesions at the end of five years, the remaining showing residual symptoms, such as nephritis, possibly of toxic origin, irritable bladders, pyuria, etc. In about 10 per cent. the course of the disease was apparently in nowise checked by the removal of the kidney. An extremely interesting, and rather surprising point, brought out by Crabtree's statistics, is that apparently in many instances very early cases, with comparatively slight destruction of kidney tissue, do worse than more advanced cases, with more extensive disease. A possible explanation for this apparently anomalous condition is offered in an introduction by Cabot, on the ground that it may "depend on at least two conditions: (1) that the renal tuberculosis is only an evidence of a generally low resistance to the tubercle bacillus, and that the failure of nephrectomy to control the process proved nothing except that it could not have been controlled by any method; (2) that the process in the kidney is so early that there has developed no immunity, and that if left to itself for a short time, such heightened resistance might come. . . . While it would seem a pity to do anything which remotely tended to discourage the only just developing enthusiasm for removing the tuberculous kidney at the earliest possible moment, it is clearly necessary to call attention at the present time to what our experience has led us to believe to be a not inconsiderable danger, viz., the lack of resistance in patients with early tuberculous

lesions of the kidney." Another interesting point brought out, is that in about 75 per cent. of cases sinuses develop, irrespective of whether the wound is drained or closed tight, the sinus development in the latter class occurring rather late, however, after apparent healing, so that had not a follow-up system been employed, the apparent results of this technic would have been much better than was actually the case. Secondary complications, especially involvement of the genital tract, were much less frequent in women than in men, and the prognosis in the former therefore considerably better.

Fatal Mercury Poisoning from Vaginal Douche.—A rather remarkable case, in which death resulted apparently from the systemic effects of mercury absorbed through the vaginal mucosa following the employment of a strong solution as a douche, is reported by FOSKETT (*Am. Jour. Obst.*, 1915, lxii, 639). The patient was an unmarried girl of twenty-two years, who took a douche consisting of three of the ordinary 7.3 grains bichloride tablets dissolved in a cup of water, and administered by means of a whirling spray. She felt a burning sensation in the vagina at once, accompanied by general distress. By the third day her condition was so serious that she was removed to a hospital. At this time the vagina and vulva were covered by a white sloughing surface; salivation was present, and the mucous membranes of the mouth and pharynx were also white and sloughy. Urinary output was as low as from 1 to 2 ounces per twenty-four hours for three days, after which it gradually came up. The urine contained albumin, granular and hyaline casts, but no mercury. On the seventh day the vaginal slough separated, followed by some bleeding. Soon after this hiccoughs and vomiting appeared, followed by bleeding from the nose and mouth, with sore throat, restlessness, and mental disturbances. The general condition grew steadily worse, incontinence developed, with blood in vomitus and stools. Death occurred on the twelfth day.

HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

MILTON J. ROSENAU, M.D.,

PROFESSOR OF PREVENTIVE MEDICINE AND HYGIENE, HARVARD MEDICAL SCHOOL, BOSTON, MASSACHUSETTS,

AND

JOHN F. ANDERSON, M.D.,

FORMERLY DIRECTOR OF THE HYGIENIC LABORATORY, PUBLIC HEALTH SERVICE, WASHINGTON, D.C.

Some Results of the First Year's Work of the New York State Commission of Ventilation.—A preliminary report of the Commission has been published by C. E. A. Winslow (chairman), D. D. Kimball, Frederic S. Lee, J. A. Miller, Earle B. Phelps, E. L. Thorndike, members

conditions gave no positive results. *Special physiological studies:* The different room temperatures were found to have no effect on the size of the dead space in the lungs, or on the acidosis of the blood, respiratory quotient, rate of digestion, heat production, creatinin in the urine, freezing-point, or specific gravity of the urine, showing that profound metabolic changes did not take place. *The quantity and quality of intellectual products and the inclination to do mental work* were studied by standardized psychological tests and by mental tests in which the subjects could either work or idle. Neither the amount or quality of the work accomplished or the desire to do the mental work were markedly affected by the different temperatures. *Inclination to do physical work:* When the subjects were required to ride a bicycle ergometer or when they had the option of working with dumb-bells for pay or idling they accomplished 15 per cent. more work in the fall at 68° as compared with 75° and in the summer 37 per cent. more work at 68° than at 86°, although the ability to work under pressure was not affected. *Appetite:* The appetite was slightly affected by temperature. In two separate series 15 per cent. more food was consumed at 68° than at 75° and 5 per cent. more at 68° than at 86°. *Comfort:* The votes as to comfort, which were based on a scale of five conditions, showed that in general the subjects were more comfortable at 68° and 75° than at 86°. *Effect of carbon dioxide and stagnant air:* In five series of experiments using a total of thirty-six subjects over eighty-five days, a comparison of the effect of breathing fresh air and stagnant air gave practically negative results. At the end of the stagnant days the average CO_2 proportion in the air was 37.9 parts per 10,000; on fresh air days seven parts per 10,000. All of the physiological and psychological responses mentioned above were found to be uninfluenced by the chemical purity of the air except the appetite. In one test 13 per cent. more food was consumed at 68° than at 86°, and in another 8.6 per cent. more at 68° than at 75°. This indication of a positive result is not entirely borne out in additional experiments now under way. The broad, tentative conclusions of the Commission based on this first year's work are: high temperatures, such as 86° and 75°, cause an increase in body temperature, in the reclining heart rate, in the excess of standing over reclining heart rate, and a slight diminution in systolic blood-pressure, and a fall in vasomotor tone but no fundamental metabolic changes. Desire for physical work is decreased at high temperatures, but mental ability is unaffected. Stagnant air at the same temperature as fresh air and without a disagreeable odor has no effect on the mental or physiological functions of the body except perhaps on the appetite for food.

State Control of Sewerage Systems.—According to the U. S. Public Health Report for October 29, 1915, the Supreme Court of Maryland has held that the section of the act of the Maryland Legislature, passed in 1914, giving the State Board of Health authority over the installation of water and sewerage systems, was not unconstitutional. The act empowered the State Board of Health to order the local authorities to install public systems of water supply, sewerage, or refuse disposal, in any county, municipality, district, subdivision, or locality where the absence or incompleteness of such systems were, in the opinion of

of the Commission, and G. T. Palmer, chief of the investigating staff (*Amer. Jour. Public Health*, February, 1915). Two problems studied by the Commission are reported on: (1) results obtained in regard to the effect of temperature and humidity upon physical and mental condition and efficiency, and (2) results in regard to the effect of carbon dioxide and organic matter in stagnant air on both physical and mental condition and efficiency. The experimental work was carried out in specially equipped ventilation laboratories at the College of the City of New York. Ninety-three paid college students acted as experimental subjects. These were observed in the room over four- to eight-hour periods during the day in seven different series of experiments each of which lasted from one to eight weeks. The effects of temperature conditions of 86° with 80 per cent. relative humidity, and of 75°, 50 per cent., and 68°, with 50 per cent. relative humidity were compared. The effects of carbon dioxide and stagnant air as compared with fresh air were studied by keeping the temperature and humidity constant and supplying no air at all or 45 cubic feet per minute per person. *Effect of environmental temperature:* The bodily responses observed and the results obtained are here given: (1) *Body Temperature:* Room temperature of 68°, 75° and 86°, have slight but definite effects on the rectal temperature. The average of 284 readings, taken at the end of a four- to eight-hour stay in the observation room at 68° was 36.73° (range, 36.50° to 39.96°), the drop from initial temperature being 0.19° to 0.80°. (Room temperature, T; body temperature, C). Similarly, 119 final readings at 75° gave an average of 36.99°. (range 36.58° to 37.10°), a fall in body temperature in four series of 0.08° to 0.44°, or a rise, obtained in one series, of 0.19°. One hundred and ninety-nine final observations at 86° averaged 37.41 (range 37.08° to 37.56°). In two series there was a rise of 0.15° to 0.43° from the initial temperature and in three a fall of 0.01 to 0.05°. *Pulse:* Exposure to a 68° temperature for four to eight hours caused an average fall in pulse rate of 11 to 13 beats. A 75° or 86° temperature gave a drop in three series of 2 to 5 beats, and in 3 cases an increase of 1 to 4 beats. The average for all observations shows that the pulse under the 86° condition was 8 beats per minute higher than under 68°, which was 66. *Increase in heart rate on standing:* The average increase in heart rate on going from the reclining to the standing position was 10 beats per minute at 68°, 24 beats at 75°, and 25 beats at 86°. *Pulse recovery after physical work:* A slower pulse recovery after physical exertion was found to exist under a 75° condition as compared with a 68° condition. *Systolic blood-pressure:* The average of all blood-pressure readings taken by the auscultatory method at the end of the period in the chamber was 116 mm. for the 68° and 75° and 112 mm. for 86°. *Crampton value:* The Crampton index of vasomotor efficiency is based on the relation between rise in pulse and change in blood-pressure on going from a reclining to a standing position. Using an arbitrary scale in which a high value is given to an increase in blood-pressure accompanied by only a slight increase in heart rate the Crampton values were 60, 45, 40, 35 as a result of staying in the room under a 68°, 75°, 86° (with fans), and 86° condition respectively. The highest vasomotor efficiency existed at a low temperature. *Respiration:* One minute respiration counts which were made under the different temperature

conditions gave no positive results. *Special physiological studies:* The different room temperatures were found to have no effect on the size of the dead space in the lungs, or on the acidosis of the blood, respiratory quotient, rate of digestion, heat production, creatinin in the urine, freezing-point, or specific gravity of the urine, showing that profound metabolic changes did not take place. *The quantity and quality of intellectual products and the inclination to do mental work* were studied by standardized psychological tests and by mental tests in which the subjects could either work or idle. Neither the amount or quality of the work accomplished or the desire to do the mental work were markedly affected by the different temperatures. *Inclination to do physical work:* When the subjects were required to ride a bicycle ergometer or when they had the option of working with dumb-bells for pay or idling they accomplished 15 per cent. more work in the fall at 68° as compared with 75° and in the summer 37 per cent. more work at 68° than at 86°; although the ability to work under pressure was not affected. *Appetite:* The appetite was slightly affected by temperature. In two separate series 1.5 per cent. more food was consumed at 68° than at 75° and 5 per cent. more at 68° than at 86°. *Comfort:* The votes as to comfort, which were based on a scale of five conditions, showed that in general the subjects were more comfortable at 68° and 75° than at 86°. *Effect of carbon dioxide and stagnant air:* In five series of experiments using a total of thirty-six subjects over eighty-five days, a comparison of the effect of breathing fresh air and stagnant air gave practically negative results. At the end of the stagnant days the average CO_2 proportion in the air was 37.9 parts per 10,000; on fresh air days seven parts per 10,000. All of the physiological and psychological responses mentioned above were found to be uninfluenced by the chemical purity of the air except the appetite. In one test 13 per cent. more food was consumed at 68° than at 86°, and in another 8.6 per cent. more at 68° than at 75°. This indication of a positive result is not entirely borne out in additional experiments now under way. The broad, tentative conclusions of the Commission based on this first year's work are: high temperatures, such as 86° and 75°, cause an increase in body temperature, in the reclining heart rate, in the excess of standing over reclining heart rate, and a slight diminution in systolic blood-pressure, and a fall in vasomotor tone but no fundamental metabolic changes. Desire for physical work is decreased at high temperatures, but mental ability is unaffected. Stagnant air at the same temperature as fresh air and without a disagreeable odor has no effect on the mental or physiological functions of the body except perhaps on the appetite for food.

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the State Board of Health, sufficiently prejudicial to the health and comfort of the community. The act further provided that local authorities, if they deemed any such order unlawful, unreasonable or unnecessary, might bring action in court to set aside the order. It seems that the State Board of Health in 1914 ordered the county commissioners of Baltimore County to install a sewerage system in a certain section of that county, stating that the absence of such a system was a menace to the health of the people. The commission brought suit to have the order set aside, and attacked the constitutionality of part of the law, but did not claim that the order was unreasonable.

PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF

JOHN McCRAE, M.D., M.R.C.P.,

LECTURER ON PATHOLOGY AND CLINICAL MEDICINE, MC GILL UNIVERSITY, MONTREAL; SOME TIME PROFESSOR OF PATHOLOGY IN THE UNIVERSITY OF VERMONT, BURLINGTON, VERMONT; SENIOR ASSISTANT PHYSICIAN, ROYAL VICTORIA HOSPITAL, MONTREAL,

AND

OSKAR KLOTZ, M.D., C.M.,

PROFESSOR OF PATHOLOGY AND BACTERIOLOGY, UNIVERSITY OF PITTSBURGH, PITTSBURGH, PA.

Experimental Studies on Adrenal.—A series of studies was undertaken by CROWE and WISLOCKI (*Bruns's Beitrage*, 1914, xcv, 8) on dogs. The adrenal is a vital organ for these animals and it would appear that the cortex is more important than the medulla. After partial extirpation the remaining portion undergoes hypertrophy, which is mainly confined to the zona fasciculata. The medulla shows no hypertrophy. After an almost complete bilateral extirpation the animals frequently have convulsions and subnormal temperature. In a few instances the animals recover without evidence of ill effect and absence of disturbances of growth or sexual function. A transient glycosuria is commonly observed in the experiment. There appears to be some relation between the adrenals and the lymphatic system. Occasionally there is a hyperplasia of the thymus. Acute general infection may cause focal necrosis in the cortex.

Studies of the Pathology of Osteogenesis Imperfecta.—The bone affections in rickets, osteomalacia, osteoporosis, scurvy and osteogenesis imperfecta are more or less related inasfar as each is dependent upon metabolic disturbances. The development of characteristics of one or other type is in part dependent upon a predisposition as well as the peculiar metabolic derangement presenting. HART (*Ziegler's Beitrage*, 1915, lix, 207) reported a case which he found considerable difficulty in classifying. A boy, aged twelve years, had, during five previous

years, taken quantities of alcohol and developed glycosuria and obesity with furunculosis. At the age of nine years atrophy and fragility of the bones developed to the degree that great deformity of the vertebræ and ribs appeared. Numerous fractures of the long bones occurred with favorable callous formation. Microscopically there was an absence in the callous of proper bone formation and osteoblasts did not appear to actively participate in the reaction. An irregular calcareous deposition took place in the injured areas. The author looks upon this case as one of osteogenesis imperfecta in which osteomalacia could not be entirely ruled out. The author suggests that in part the disturbance of the bone tissue was brought about through the unusual development of fatty tissues in the bones. There was no evidence that the endocrin glands were at fault. The author believed that this individual was possessed of an abnormal constitution whose peculiarities were only demonstrated through the influence of alcohol. Thus osteogenesis imperfecta need not always appear as a congenital disease, but its predisposition may only manifest itself in the presence of particular constitutional derangements in postnatal life.

Histological and Experimental Observations on the Destruction of Tumor Cells in the Bloodvessels.—Various authors have studied the fate of tumor cells in different tissues. At times, individual tumor cells are destroyed by the phagocytosis of giant cells, at other times a lytic destruction leads to a spontaneous disappearance of small tumor masses. Such methods of tumor cell destruction are now well recognized so that it is fully appreciated that each tumor cell liberated from the main mass does not necessarily survive. IWASAKI (*Jour. Path. and Bacteriol.*, 1915, xx, 85) studied a number of human malignant tumors including cancer, sarcoma, and hypernephroma, observing particularly the character of tumor emboli. These emboli are either naked, covered with recent clot or show an advancing organization of the blood clot around them. Within each type various grades of necrosis beginning in the centres were found. The destruction of the tumor cells occurs either through want of nutrition or by the unfavorable character of the granulation tissue. Nevertheless, tumor masses may remain latent for long periods of time. The author also carried out a series of experiments in which the Jensen tumor was inoculated intravenously with mice. The vascular emboli were subsequently studied and in general their fate was similar to that of the human. The author believes that there is a special affinity between tumor cells and the cells of certain organs.

Contributions to the Pathology of Lipomatosis.—One of the most common disturbances of tissue metabolism is found in the varying accumulation of fat in the interstitium. MATSUOKA (*Jour. Path. and Bacteriol.*, 1915, xx, 106) studied the development of fat tissue under a variety of conditions from infancy to old age. It is readily appreciated that no definite division between the physiological and pathological deposits of fat tissue can be determined. The lipomatosis may be general or localized. Under some conditions lipomatosis appears to be a progressive process whereby the fatty tissue is superimposed upon the tissues of an organ. On the other hand, lipomatosis also develops

in consequence to an atrophic process. In the latter, the primary atrophy is usually associated with fibrosis in which the fatty tissue appears to take its origin. The author believes that the fat cells are nothing more than modified connective-tissue cells. He, however, offers no evidence for these conclusions. During the development of local or general lipomatoses the involved area shows an unusual hyperemia

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Atrophy in Adipose Tissue and the Histogenesis of Fat Cells.—MATSUOKA (*Jour. Path. and Bacteriol.*, 1915, xx, 118) claims that atrophy of adipose tissue is common, and that one may quite readily distinguish the degree of atrophy by the contour of the shrinking cells. Fat atrophy is commonly accompanied by an increase in the connective tissues. The early stages, however, show a serous infiltration. This infiltration is made possible through the great vascularity of adipose tissue. In the proliferation of fat cells "the old fat drop becomes absorbed and deposited in the young cell." The nucleus shows a definite activity in the metabolism of fat and in true fat cells globules of sudan-staining material are present in the nucleus. The lobulated adipose tissues can be converted into bundles of spindle-shaped connective-tissue cells, and for example, the whole epicardial fat which is derived from fibrous tissue, can be reconverted into such connective tissue. Thus fat cells of every kind result from a metaplasia of connective-tissue cells.

Chronic Interstitial Nephritis and Tuberculosis.—The influence of tuberculosis in leading to a chronic interstitial nephritis has been suggested by a number of observers. SCHOENBURG (*Virchows Archiv*, 1916, ccxx, 285) reports a second series of cases supporting his previous view on the importance of tuberculosis in bringing about chronic fibrotic processes in the kidney. These lesions are seen at all periods of life, but the more interesting ones occur in the second and third decade where a histological analysis more clearly demonstrates the origin of the process. The kidney lesion may appear in the form of isolated scars or diffuse fibrosis. The former are the result of tuberculous processes about the arteries whereby circulatory disturbances lead to hyaline degeneration of the glomeruli and atrophy of tubules of cortex. This type of lesion simulates the arteriosclerotic kidney. In the second type the process is more diffuse and the fibrous tissue is more evenly distributed. Here the glomeruli are less involved while a general atrophy of the cortical tubules is found. Commonly the two conditions are seen in the same kidney.

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